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THE PHYSICAL SIGNS OF PULMONARY DISEASE.

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THE

PHYSICAL SIGNS OF PULMONARY DISEASE

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THE

PHYSICAL SIGNS

OF

PULMONARY DISEASE

BY

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TO THE

STUDENTS OF THE MANCHESTER ROYAL INFIRMARY

FOR WHOM IT WAS WRITTEN

I DEDICATE

THIS LITTLE WORK

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ANATOMICAL INTRODUCTION.

In form each lung is somewhat conical, and possesses an apex, a base, outer and inner surfaces, and anterior, posterior, and inferior borders. The base of the lung is that part of the surface which rests upon the diaphragm. It is concave, to correspond with the convexity of the latter. The apex is rounded, and extends from an inch to an inch and a half above the first rib (the right being usually somewhat the higher). The external surface is convex, and in contact with the thoracic wall. The internal surface is concave, and embraces the pericardium. The bronchi and large vessels forming the root of the lung enter this surface near the posterior border. The anterior border is thin, and intervenes between the pericardium and anterior wall of the chest. The posterior border, on the contrary, is thick and rounded, and occupies the concavity formed by the bodies of the vertebræ and the ribs. This border is much longer than the anterior. The inferior border bounds the concave base of the lung, and lies between the diaphragm and ribs. Each lung is surrounded by a pleural sac, with its potential cavity. The lung is divided into lobes by a fissure which, commencing posteriorly about three inches from the apex, passes downwards and forwards to reach the anterior border. In front this fissure is found on a level with the sixth interspace. The superior lobe of the right lung is further divided by a fissure running nearly horizontally from the anterior border to meet the fissure just described. This horizontal fissure is situated about the level of the third space.

In conformity with the greater projection of the heart to the left, and the greater height and convexity of the right half of the

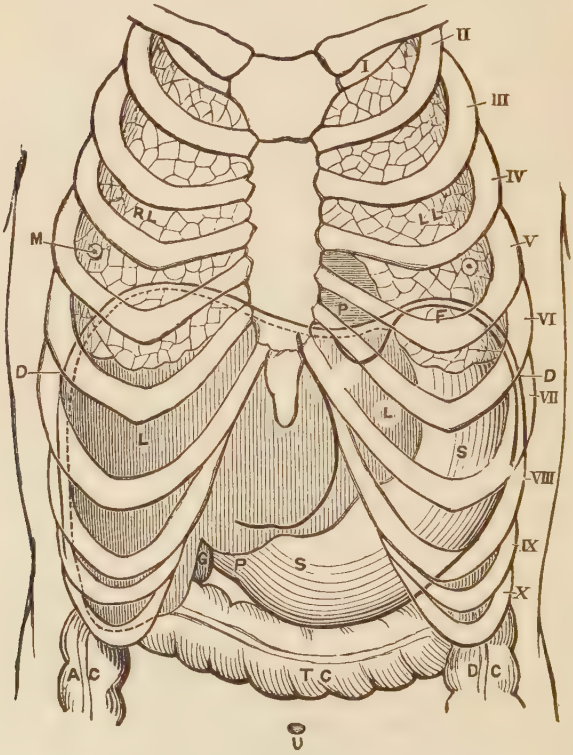

Fig. I. (from Professor Turner's *Anatomy.*)—The Lungs, and their relations to the diaphragm, liver, and stomach. II to X, second to tenth ribs; DD, diaphragm; RL, right, and LL, left lung; P, pericardium; SS, stomach; F is placed superficial to its fundus; P, its pylorus; C is over the cardiac orifice; L L, the liver; G, gall bladder; AC, ascending, TC, transverse, and DC, descending colon; U, umbilicus; M, right nipple. Modified from Luschka.

diaphragm due to the liver, the left lung is narrower and longer than the right.

Fig. III. represents the tubular apparatus which traverses

the pulmonary substance, and forms communication between its vesicles and the external air. Commencing at the larynx on a level with the body of the sixth cervical vertebra, the tracheal portion extends down to the body of the fourth dorsal vertebra. The trachea then divides into the right and left bronchi, the right being more horizontal, shorter, and of somewhat greater calibre. Having reached the internal surface

Fig. II. (from Professor Turner's *Anatomy.*)—The Vesicular Structure of the Lung. B, lobular bronchus; *av*, air vesicles; A, pulmonary artery; *c*, pulmonary capillaries V, pulmonary vein.

of the lung and plunged into its substance, the bronchi divide and sub-divide; the tubes resulting from this division gradually diminish in size, but they retain supporting cartilaginous structure in their wall until they reach a diameter of about the $\frac{1}{20}$th of an inch, although this in its amount and regularity of distribution decreases steadily from the entrance of the bronchi into the lung.

Artificial Division of the Chest into Regions.—To facilitate the

localisation of physical signs, the anterior, posterior, and two lateral aspects of the thorax have been respectively divided artificially into regions.

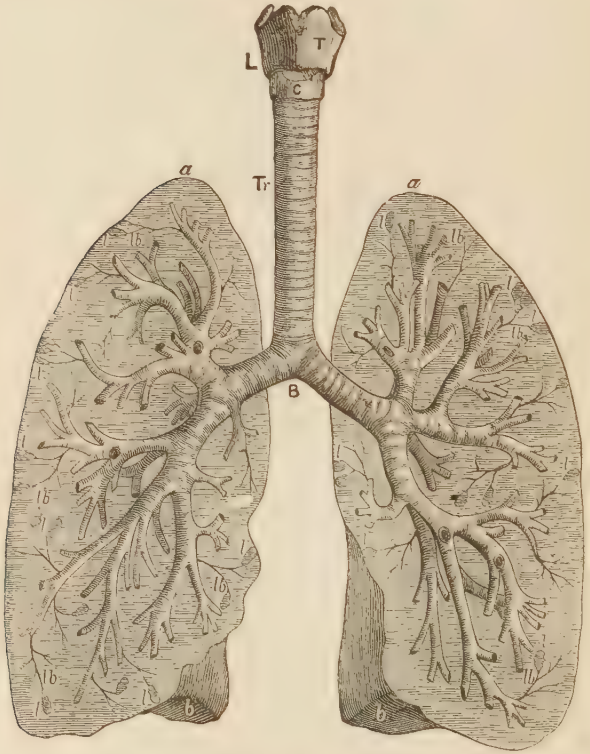

Fig. III. (from Professor Turner's *Anatomy*.)—The Windpipe and Lungs. L, larynx; T, thyroid cartilage; C, cricoid; *Tr*, trachea; B, bifurcation into bronchi; *a*, apex, and *b*, base of each lung. The ramifications of the bronchi within the lungs are shown; *lb lb*, lobular bronchial tubes; *l*, *l*, *l*, lobules.

Anteriorly, the surface is mapped out into thirteen regions, which are as follows:—

(*a*) A median and single group.
(1) Supra-sternal.
(2) Superior sternal.
(3) Inferior sternal.
(*b*) A lateral and double group.
(1) Supra-clavicular.
(2) Clavicular.
(3) Infra-clavicular.
(4) Mammary.
(5) Infra-mammary.

(*a*) (1) The supra-sternal region or notch, as it is commonly called, is bounded laterally by the sterno-mastoid muscles, and corresponds chiefly to the trachea. (2) The superior, and (3) inferior sternal regions are divided by a line joining the lower borders of the third costal cartilages, and are bounded by lines representing the borders of the sternum.

(*b*) The regions belonging to this group are of course double, one existing on each side. (1) The supra-clavicular region is bounded below by the inner half of the clavicle, on the inner side by the trachea, and superiorly by an imaginary line drawn from the middle of the clavicle to the upper rings of the trachea. (2) The clavicular region corresponds to the inner half of the clavicle. (3) The infra-clavicular region extends down from the clavicle to the lower border of the third rib, and is bounded internally by the border of the sternum, and externally by a line drawn vertically downwards from the middle of the clavicle. (4) The mammary region is bounded above by the last region, internally by the sternal border, and externally by a vertical line drawn downwards from the middle of the clavicle, and inferiorly by the sixth rib. (5) The infra-mammary region is bounded superiorly by the mammary, inferiorly by the margin of the costal framework, and externally by the line limiting in the same direction the mammary region. This region lies below the normal lung border.

The lateral aspects of the chest are each divided into two regions :—

(1) Axillary
and
(2) Infra-axillary.

(1) The axillary region extends from the apex of the axilla down to the sixth rib, and is bounded anteriorly by the anterior regions of the same level, and posteriorly by the inferior border of the scapula. The latter is, of course, a movable boundary. (2) The infra-axillary region extends downwards from the last region to the costal margin, and is bounded anteriorly by the infra-mammary region, and posteriorly by a vertical line drawn from the inferior angle of the scapula.

The scapulæ are made the basis of the division of the posterior aspect of the chest into regions, which are, on each side, as follows :—

1. Supra-scapular.
2. Supra-spinous.
3. Infra-spinous.
4. Inter-scapular.
5. Infra-scapular.

The scapular region is co-extensive with the scapula, and is subdivided by the spine of that bone into supra-spinous and infra-spinous regions. The small portion of chest wall above the scapula is sometimes termed supra-scapular. Between each scapula and the spine we have an inter-scapular region. Inferiorly on each side there is the infra-scapular region, bounded above by a line drawn transversely from the inferior angle of the scapula to the corresponding dorsal spine (generally the seventh), below by the twelfth rib, externally by the infra-axillary region, and internally by the spine.

To recapitulate, the artificial regions into which the thoracic surface is divided are as follows :—

Anterior Aspect.

1. Supra-clavicular.
2. Clavicular.
3. Infra-clavicular.
4. Mammary.
5. Infra-mammary.
6. Supra-sternal.
7. Upper-sternal.
8. Lower-sternal.
9. Supra-clavicular.
10. Clavicular.
11. Infra-clavicular.
12. Mammary.
13. Infra-mammary.

Lateral Aspects.

14. Axillary.	16. Axillary.
15. Infra-axillary.	17. Infra-axillary.

Posterior Aspect.

18. Supra-scapular.		23. Supra-scapular.	
19. Supra-spinous.	Scapular.	24. Supra-spinous.	Scapular.
20. Infra-spinous.		25. Infra-spinous.	
21. Inter-scapular.		26. Inter-scapular.	
22. Infra-scapular.		27. Infra-scapular.	

Still further to promote precision in the localisation of morbid phenomena, certain imaginary vertical lines are described. Thus, a line drawn through the middle of the sternum is spoken of as the median line; one drawn through the nipple, as the mammary; one drawn midway between the sternal border and the nipple, as the parasternal; one drawn downwards from the apex of the axilla, as the axillary; and one from the angle of the scapula, as the dorsal line.

INSPECTION.

ABNORMITIES IN THE SHAPE OF THE CHEST.

A.—Bilateral and Symmetrical.

1. Developmental Abnormities:—
   - (*a*) The Alar Chest.
   - (*b*) The Flat Chest.
   - (*c*) The Pigeon Chest.
   - (*d*) The Rickety Chest.
2. Abnormities due to Emphysema.

B.—Unilateral Irregularity of Form.

- (*a*) Expansion.
- (*b*) Retraction.

C.—Local Irregularity of Form.

- (*a*) Bulging.
- (*b*) Depression.

A.—Bilateral and Symmetrical Abnormities in the Shape of the Chest.

1. *Developmental Abnormities.*—(*a*) The Alar Chest is characterised by its small transverse sectional area, brought about chiefly by exaggerated obliquity of the ribs. For the same reason, it is elongated vertically. The shoulders droop, and the inferior angles of the scapulæ project from the trunk, giving origin to the name.

(*b*) Another variety of small chest—the Flat Chest—differs from the last described, which showed little departure from the normal, as regards the shape of its transverse sectional area, by being flattened from before backwards, the anterior boundary losing its curve and becoming straight. This form of chest is not necessarily accompanied by the exaggerated obliquity of the ribs distinguishing the alar chest.

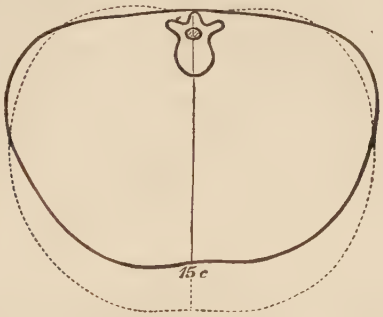

Fig. IV, (From Dr. Gee's *Auscultation and Percussion*).—Circumference = 56·5 centimeters.

ALAR THORAX.

Tracing taken from a child. The dotted line indicates the shape of the chest of a healthy child of the same size. The circumferential measurement refers to the alar thorax.

These abnormal types are commonly, though not necessarily, combined. Both indicate a deficient capacity for lung, and this result is still further accomplished by the frequent presence of a transverse sulcus running outwards and somewhat downwards from the xiphoid cartilage on both sides, to cease about the mid-axillary line. This depression in early life marked the upper border of the abdominal organs, which, as Dr. Sibson wrote, "in children are of greater bulk than the thoracic; and when, owing to the descent of the diaphragm, the latter replace the former, the walls of the chest collapse wherever the smaller thoracic replace the larger abdominal organs." * Persons with

* *Medico-Chirurgical Transactions*, vol. xxxi. p. 375.

chests possessing the characters described are usually regarded as being predisposed to phthisis, and the belief seems well founded, although many such individuals escape. It will be noticed, that in these small chests the antero-posterior diameter is especially diminished; while we shall see that in diseases accompanied by increase of the lung space, this diameter is again chiefly affected, though in the opposite direction.

The morbid types that remain to be considered differ from the foregoing, inasmuch as in place of shadowing forth possible future disease, they bear the record of past ill-health.

(*c*) The Pigeon Chest.—In this, as the cyrtometrical tracing renders evident, the transverse area of the chest at the level of

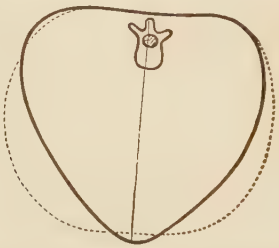

Fig. V. (Dr. Gee).—Circumference = 57·5 centimeters.

PIGEON BREAST.

Tracing taken from a child of seven years. Dotted line indicates natural shape at same age.

the xipho-sternal articulation assumes the triangular form. The mode of production of this abnormity will engage our attention later on; here suffice it to say that it is the result of obstruction to the entrance of air during strong inspiratory efforts, when the parietes possess little power of resistance to the atmospheric pressure.

(*d*) The last abnormity of this class we have to consider, is the Rickety Chest. This somewhat resembles the pigeon chest, but reference to diagram VI. will show that it is produced by a special yielding at one point of the circumference, namely, the costo-chondral articulation. A longitudinal groove is thus formed

on each side in the position indicated. It is probable that this abnormity may result apart altogether from the existence of obstruction to inspiration in the air passages, the softening of the skeleton at the affected points sufficiently accounting for the malformation.

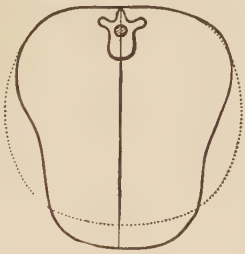

Fig. VI. (Dr. Gee).—Circumference = 42·75.
Rickety Chest.
Dotted line indicates shape of chest in an infant of about the same age.

2. *Abnormities due to Emphysema.*—The altered form of the thorax in extensive pulmonary emphysema is one of the most striking features in the physiognomy of the disease. The chest becomes enlarged and more or less barrel-shaped, the sternum is projected forwards, and frequently arched from above downwards, the ribs have lost their normal obliquity, the intercostal spaces are widened, and the spine is unduly curved. The antero-posterior diameter undergoes the greatest increase, and may actually exceed the transverse diameter. In many cases the lower zone of the chest is relatively retracted. To enter into a description of the anatomical changes in the lungs which underlie the alterations of thoracic form we have just described, is beyond the scope of our subject, but some reference to the mode of production of emphysema seems necessary.

For the clinical observer, emphysema may be said to consist of dilatation of the pulmonary vesicles, with loss of elasticity in the structure constituting their walls, and the more or less constant presence of bronchitis. The lungs are increased in volume, hence the enlargement of the thorax. The distending force at

work may be applied during inspiration or expiration, or both. Even when applied during inspiration, departure from the normal occurs first with expiration. The inspiratory expansion of the chest being muscular and the expiratory depression the product of elastic recoil chiefly, agents which diminish the elasticity of the lungs and that of the bony and cartilaginous thoracic framework, without materially interfering with the muscles, will

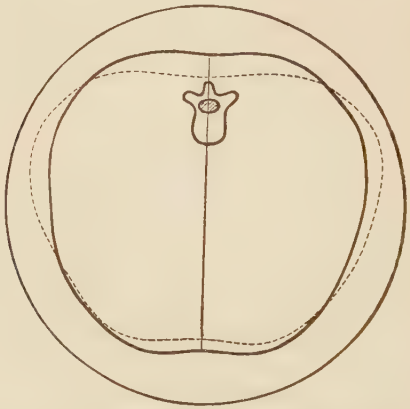

Fig. VII. (Dr. Gee).—Bilateral Enlargement of Emphysema.

Inner line=emphysematous chest.
Outer line=a circle drawn to show how nearly the emphysematous approaches the circular shape.
Dotted line=natural adult chest.
Actual measurements in centimeters:

Circumference	= nat.	89·	emphys.	87·75
Transverse	= ,,	29·6	,,	27·25
Antero-posterior	= ,,	22·25	,,	25·4

render expiration imperfect, so that inspiration is made habitually with an over-full chest; and in order that sufficient tidal movement of air may be accomplished, expansion must be carried, as it were, to a higher level; in other words, the lungs become over-distended.

This consideration is also to some extent applicable to cases where expiration is impeded by obstruction in the tubes. It also accounts for the emphysematous tendencies of old age.

To Sir William Jenner* we mainly owe the elucidation of the production of the emphysematous lung, and the consequent abnormities in the shape of the chest. He thus enumerates "the conditions essential to the over-distension of the air vesicles.

"(*a*) Inflation of the lung.

"(*b*) Closure of the natural passage for the escape of air from the lung.

"(*c*) Unequal pressure on the lung.

"(*d*) Unequal support of different parts of the lung." He goes on to state that "during violent cough and great muscular effort, these essential conditions are fulfilled :—

"(*a*) Preparatory to cough and to great muscular effort, a deep inspiration is taken, *i.e.*, the lungs are inflated.

"(*b*) Then the glottis is closed, *i.e.*, the air is prevented escaping by the natural channel.

"(*c*) Then, by the action of the expiratory muscles, the lungs are strongly compressed, and an examination of the structure of the thoracic walls at once shows that the compression must be unequal.

"(*d*) Examination of the structure of the walls of the chest also shows that the support offered to the lungs by these walls is very different in degree at different parts."

With reference to (*c*) and (*d*) after describing the movements which must take place between the lung surface and chest wall, the author remarks :—"By these changes in the lungs and in the chest-walls, their relative positions are being constantly brought to correspond to the ribs and to the intercostal spaces, &c., and thus ultimately the air vesicles of the whole lung may be over-distended."

The "permanence-securing causes" of the distension so brought about do not here concern us. Sir William Jenner's theory seems to be in accordance with the comparative contraction of the lower zone of the chest so commonly observed in emphysematous patients. In a healthy person, during coughing, retraction at this level of the chest occurs, and is, no

* *Reynold's System of Medicine*, vol. iii. p. 479.

doubt, due to the powerful contraction of the abdominal muscles. The lowest portions of the lungs, especially on the right side, are then well supported, during coughing.

B.—Unilateral Iregularity of Form.

(*a*) *Expansion—Pleural Effusion.*—The changes in thoracic form brought about by pleural distension demand notice first

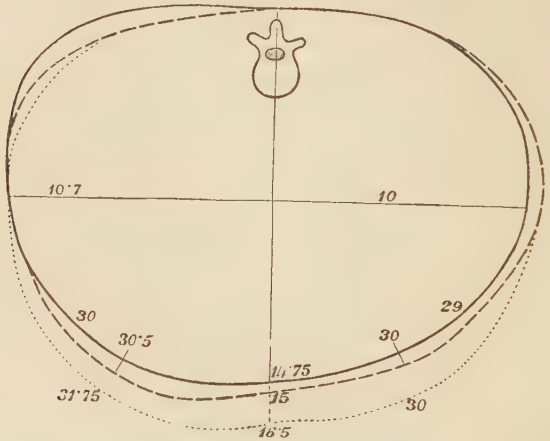

Fig. VIII. (Dr. Gee).—Unilateral enlargement of chest (right side); artificially produced by injecting air into the right pleural cavity.
Unbroken line = outline before injection.
Broken line = outline after moderate distension.
Dotted line = outline after extreme distension.

Figures at bottom of vertical line indicate the antero-posterior diameters; along horizontal line indicate transverse semidiameters; remaining figures indicate right and left semicircumferences.

of all, in this section, inasmuch as they possess some degree of right to be included in the last. Our meaning will be best explained by reference to Fig. VIII. It represents by means of a cyrtometer tracing the alterations in form undergone by the chest during the artificial distension of the right pleura with air.

We see that the changes produced are by no means unilateral only, and that the whole circumference takes part in the change.

Notwithstanding the eye directed upon the chest of a patient with distended pleura detects unilateral change almost alone, and this no doubt chiefly because of the difference of movement on the two sides, and to the obliteration of the intercostal depressions.

A very little consideration will show that the changes indicated by the cyrtometer are only those that might have been theoretically anticipated. The contents of the chest are to be regarded as one whole, and not as made up of halves divided by an unyielding partition. Centrifugal pressure arising, then, in one half, and of course extending in every direction, being due to the accumulation of fluid, will bear on the other half of the contents as well as on the parieties of its own side. Respiration must be carried on, and the space of the healthy lung being encroached upon by the heart and mediastinum, the patient expands his chest to the utmost, and in this way endeavours to make room for the lung which is still active. Consequently, the thorax is enlarged as a whole, and, as in emphysema, it is the antero-posterior diameter which is most increased; but inasmuch as the intra-thoracic expanding force is exerted directly on the side of effusion, and only indirectly on the healthy side, the enlargement takes place in somewhat greater degree on the former. For clinical purposes, the most valuable measurement, as an indication of unilateral enlargement, is that of the vertebro-mammary diameter, easily made by compasses.

In copious pleuritic effusion, and especially in empyema, obliteration of the intercostal depressions and widening of the interspaces is easily detected by the eye. The intercostal spaces may bulge, but this latter phenomenon only occurs where the muscular planes are themselves paralysed as an effect of contiguous inflammation, and therefore in empyema. The angle formed by the costal margin with the median line of the abdomen is enlarged. In pneumothorax the affected side assumes the dimensions of the chest on forced inspiration, and extreme distension results only from the presence of fluid effusion *plus* the air. Accumulation of air in the pleura, with

high tension of the wall, depends upon the free entrance and deficient exit of air, during forced thorax movements, the necessary result of dyspnœa, and is brought about by the fistulous communication between the lung and pleura acting after the manner of a valve. When pneumonia affects a whole lung, the affected side remains in the state of inspiratory expansion; but this enlargement cannot he recognised by the eye. Malignant growths of the lung may, however, produce enlargement of the side, closely simulating that due to pleuritic effusion.

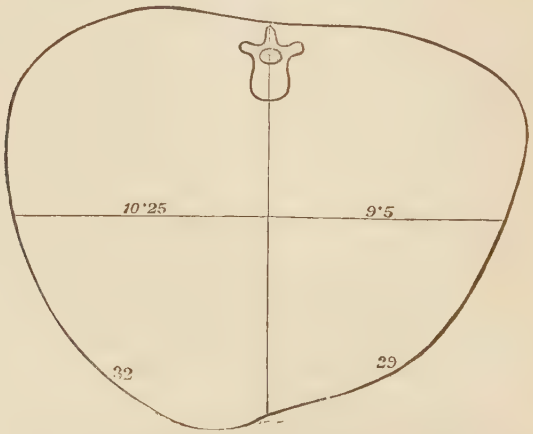

Fig. IX. (Dr. Gee).—Unilateral retraction of chest: consequent upon cirrhosis of left lung in a girl of fourteen.

(*b*) *Retraction.*—Unilateral retraction of the chest is met with in the following diseases:—(1) Pleurisy, the fluid having been absorbed while the lung has failed to expand; (2) Cirrhosis of the lung or fibroid phthisis; (3) Collapse of a lung from pressure on its bronchus; and (4.) cancer infiltrating a whole lung. Of these the two former are comparatively common, the two latter rare.

(1) The shape assumed by the chest in the retraction period of pleurisy differs from that which occurs in the unilateral di-

minution resulting from other causes in the following respects. The spine is usually curved laterally with the concavity towards the affected side, and the corresponding shoulder is lowered, the ribs being approximated and undergoing rotation, by which their external surfaces become inferior. This rotation affects all the ribs except the lowest, but commences above.

(2) Cirrhosis of the lung produces diminution of the affected side of the thorax, but the shape remains more regular than that due to pleurisy with retraction; the curvature of the spine occurring in the former, is little marked or absent, and rotation of the ribs is not produced.

(3.) Collapse of a lung occasions a uniform diminution of the affected side, and the distortions following pleurisy are not met with.

(4.) Certain rare forms of pulmonary cancer cause retraction of the side.

C.—Local Irregularities of Form.

The description of local irregularities of form, although they are of great practical importance, need not detain us long. They mostly resolve themselves into bulging and depression of portions of the thoracic wall.

(*a*) Local Bulging, with obliteration of the intercostal depressions in the lower part of the chest, points strongly to the presence of fluid in the pleura, and is best developed in encapsuled empyema. Intra-thoracic growths are another though comparatively rare cause of local bulging. Pneumonic consolidation probably never produces it. Deformities of the spine exert a baneful influence on the shape of the chest, depressing some portions and bulging others; but the origin of such abnormities will be evident from the spinal condition.

(*b*) Infra-clavicular Depression on one or both sides (in the latter case usually unequal) forms a valuable sign of phthisis. Local depression at any part may result from the causes we have mentioned, as productive of unilateral general depression, when their influence is confined to a limited area only.

Common physiological bulgings, and depressions such as the former over the sternum, where it is joined by the second cartilages, and the latter over the same bone at the lower part must not be confounded with the alterations in form, which indicate disease.

Œdema of the subcutaneous cellular tissue of the lower part of the thorax is not unfrequently observed over pleuritic effusions, especially when these are purulent.

ABNORMITIES OF CHEST MOVEMENT.

I. Bilateral and Symmetrical.
   - (*a*) Upper and Lower Costal Types of Respiration, &c.
   - (*b*) Abnormities due to Emphysema.
   - (*c*) Inspiratory Antero-lateral Retraction of the Inferior Zone of the Chest.

II. Unilateral Abnormities of Movement.
   - (*a*) Diminution
   - (*b*) Exaggeration

   } of Movement.

III. Local Abnormities of Movement.
   - (*a*) Diminution.
   - (*b*) Movements due to Pleuritic Effusion escaped Subcutaneously.
   - (*c*) Pulsating Empyema.

I.—Bilateral and Symmetrical Abnormities.

(*a*) *Upper and Lower Costal Types of Respiration, &c.*—The act of inspiration having for its end the enlargement of the space occupied by the lungs, is accomplished by means of a centrifugal movement of the walls which bound that space, the result of muscular contraction, while the mere recoil of elastic structure in these walls and in the lungs themselves constitutes the expi-

ration of quiet breathing. Muscular contraction may, however, under certain circumstances be brought into play for the purposes of expiration, and with greater force than is available for inspiration. The alternate enlargement and diminution of the lungs which must of necessity accompany the like changes in the thoracic space they occupy, is only possible by an ebb and flow of the intrapulmonary air. In this way is respiration carried on.

It is clinically useful to consider the inspiratory movement of the chest wall as a combination of expansion and elevation, inasmuch as we find that the latter fails, while the former may continue little altered under conditions which interfere with the due entrance of air into the lung.

We have further to consider the action of the diaphragm, which by its descent during contraction enlarges the lung space downwards. Its movements necessarily occasion a protrusion and recession of the abdominal wall, which are not without significance in pulmonary diagnosis. With inspiration there is elevation of the abdominal wall, with expiration recession of the same. The ascent of the diaphragm is accomplished in quiet breathing by the elasticity of the lungs.

The sexual types of respiratory movement, which, however, hold absolutely good only in tranquil respiration, have now to be considered. The female type consists in the free movement of the upper zone of the chest, while diaphragmatic and lower costal expansion is but feebly exercised and sequentially in time. In the male type, on the other hand, quiet respiration is carried on almost entirely by the movements of the diaphragm and chest wall of the lower zone, which act in a sense antagonistically, though to the same end, namely, the enlargement of the lung space. To observe this type of respiration in perfection the patient should be seated, and care taken to direct his attention away from the object of examination. What little upper costal movement there may be, follows in time the lower expansion. In forced respiration, however, as when the patient is asked to take a deep breath, the upper costal movement not only

comes into much greater prominence, but precedes the diphragmatic, or rather *commences* the whole inspiratory expansion.* It thus approaches the female type we have already described. In all efforts to inspire, then, we may expect in the male as well as in the female a predominance of the upper costal or thoracic type of respiration. These considerations have reference chiefly to inspiration ; let us now turn to expiration. We have already stated that this, in quiet breathing, is the result of the elasticity of the chest wall and lung structure, which comes into play immediately on the cessation of the muscular contraction of inspiration. It must be remembered that muscular contraction may be brought to bear on the expulsion of air from the chest with great force, such expulsion being accomplished, of course, by a diminution of the lung space, resulting from a centripetal movement of its walls. The diaphragm is unable by virtue of its own contraction to diminish the space in an upward direction, but its ascent is effected with considerable force by contraction of the abdominal muscles, which, powerfully compressing the abdominal viscera, drives them upwards towards the thorax, thus elevating the diaphragm and diminishing the lung space. Forcible expiration is well observed in the act of coughing. Coughing is a complicated action, consisting of (1) a deep inspiration, (2) closure of the glottis, (3) powerful contraction of the expiratory thoracic and abdominal muscles, and (4) bursting open of the closed glottis with explosive violence.

During the inspiratory expansion of the chest walls in health the intercostal depressions are noticed to deepen, while in expiration they regain their former level. When inspiration is forced their subsidence is increased, while during expiration they tend to disappear, the interspaces becoming level with the ribs. This is only what one may have anticipated. The bony and

* Individual peculiarities are from time to time met with. Thus, a male patient with chronic Bright's disease, when asked to take a deep breath, expanded first of all the lower part of the lung space by combined diaphragmatic and lower costal movement, and then the upper part of the chest. If the breath was less deep, the latter movement did not take place at all.

cartilaginous framework in its centrifugal movement drags with it and inflates the lung, but the latter is ever striving by virtue of its elasticity to resist, and the resistance it offers, assisted by any obstacle that may exist to inflation, falls with greatest effect on the soft portions of the thoracic wall which relatively yield to atmospheric pressure. During expiration, again, the parietal elasticity, as it were, overtakes that of the lungs, and these organs require a certain amount of time to rid themselves of the necessary amount of air. The more the elasticity of the lung is impaired, and the greater the obstacle to the expulsion of air, the more do the intercostal depressions tend to disappear, and hence most characteristically in emphysema with bronchitis. It is probable, however, that bulging of the interspaces does not take place, while the intercostal muscles remain active. In health the lower border of the lung can be very fairly determined simply on inspection, by observing the movements of the intercostal spaces above, and their absence below the lung. This difference is more striking on the right side.

In disease the sexual types of respiratory movement may be reversed. Thus female patients, whose lungs in the upper portions are much damaged by phthisis, may breathe chiefly by means of the diaphragm and lower costal expansion. In hysteria the same change of type commonly occurs as one of the vagaries of that disease. The remarkable abdominal protrusion (capable of being persistently maintained for long periods) frequently observed in the hysterical, is probably also an abnormity of respiratory movement, inasmuch as it seems to depend upon a tonic contraction of the diaphragm. Such patients will be found to breathe in exaggerated female fashion, the lower portion of the chest being almost motionless. Tympanites no doubt plays a considerable part in many of the hysterical enlargements of the abdomen, but it seems to us that the special form of protrusion referred to is accomplished by a sustained depression of the diaphragm, and its speedy disappearance under the action of chloroform bears out this view. In choreic girls, the diaphragm frequently acts with great force,

and in sudden jerks which depress the anterior chest wall, while they protrude the abdomen.

In the male patient, on the other hand, all causes which impede the action of the diaphragm tend to render the respiratory movement, of the upper thoracic or female type. This is characteristically observed in general peritonitis, or in that limited to the surface of the diaphragm and adjoining parts. Ascitic and pericardial accumulations also interfere with the due movement of the diaphragm, and bring into prominence upper costal movement. Paralysis of the phrenic nerves, such as may occur from spinal pachymeningitis, renders the descent of the diaphragm impossible, and thoracic expansion is frequently then accompanied by recession of the abdominal wall at the epigastrium, owing to the diaphragm being drawn up, as a vacuum is created above by the centrifugal movement of the chest walls, while on their recoil apparent epigastric protrusion takes place. This is rather abdominal retraction during inspiration than protrusion during expiration. Cervical spinal pachymeningitis may also lead to another and opposite type of abnormity in respiratory movement, brought about by paralysis of the nerves given off below the level of the phrenics when respiration is carried on almost alone by the diaphragm.

(*b*) *Abnormities of Movement dependent upon Emphysema.*— The abnormity of movement characteristic of the emphysematous chest depends upon the following conditions :—

(1) The lungs *persistently* contain an amount of air greater than that contained in healthy lungs at the end of a full inspiration.

(2) The lung structure has lost its elasticity.

(3) Bronchitis is so common as to form a feature of the disease.

The first of these concerns us with regard to inspiration ; the second and third, with regard to expiration. The inspiratory movement must be made "on the top of inspiration," that is, *plus* a degree of inflation of the lungs, which in health should be *approached* only at the *end* of inspiration. We have already

referred to the fact that thoracic movement is always predominant in forced breathing, and consequently where there is dyspnœa.

The diaphragm in emphysema and bronchitis descends with force, as evidenced by protrusion of the abdominal wall; it is often unable, however, to inflate the lower portions of the lungs, which are elongated at the expense of the voluminous emphysematous lung above and antero-lateral retraction of the chest wall at the corresponding level of the thorax.* To return to the thorax, the contraction of the inspiratory muscles acting upon it is in excess if measured by the force exerted, but their energy is expended chiefly in elevating the chest as a whole, which accomplishes the introduction of only a small quantity of air. The ebb and flow of intra-pulmonary air is thus rendered defective; of stagnant air there is more than enough.

Again, two causes are at work to interfere with the due expulsion of what little air has been introduced during the inspiratory struggle. These are loss of elasticity in the lung structure and obstruction from bronchitic secretion in the tubes.† An extremely prolonged and laboured expiratory movement, in which muscular contraction plays a conspicuous part, is then a characteristic feature of pulmonary emphysema.

* "During inspiration the diaphragm descends with great force, drawing down and elongating the inferior portion of the lungs, while the upper part of the chest moves forwards and upwards, expanding the superior portion of the lungs. While the abdomen and upper part of the chest protrude, the lower end of the sternum and the adjoining costal cartilages collapse during inspiration;" . . . "the lungs are enlarged above and lengthened below more rapidly than air can enter them; and, owing to atmospheric pressure, they necessarily collapse below, and the walls of the chest then fall backwards."—Dr. Sibson "On the Movements of Respiration in Disease," *Med. Chir. Trans.*, vol. xxxi. pp. 403, 404.

† It may be asked, why obstruction in the tubes should be more effective during expiration? In emphysema expiration cannot be accomplished by the lung's own elasticity, and pressure has to be brought to bear upon the expulsion of air from outside. Such pressure will act upon the small tubes as well as upon the vesicles, and in this way the exit of air from the latter must be impeded. In inspiration, again, the small tubes will tend to widen.

The intercostal spaces in emphysema are depressed during inspiration, but become level with the ribs during expiration. It is probable that they never bulge beyond the ribs, while the intercostal muscles remain healthy. In some cases of emphysema the diaphragm lies permanently at a lower level. Dr. Stokes * spoke of "this circumstance . . . . . as a most distinguishing mark in cases of this disease, which may be divided into those with and those without diaphragmatic displacement." When depression occurs, it of course still further embarrasses respiration. It is very probable that the diaphragm becomes hypertrophied in emphysema, and some emphysematous patients with unusually rigid costal frameworks breathe almost alone by means of it.

To recapitulate. In emphysema accompanied by bronchitis the thorax is heaved up *en masse*, and the diaphragm descends, protruding the abdominal wall, while the lower ribs fall in antero-laterally from atmospheric pressure and the direct action of the diaphragm. When bronchitis passes off, the abnormity of movement last described passes off in greater or less degree. Often a slight drawing in of the lower thorax in its anterior third at the beginning of inspiration, followed immediately by fairly good expansion of the sides at the same level, is all that remains. Such retraction is probably due to the first pull of the diaphragm upon its peripheral attachments when its action is exaggerated.

(*c*) *Inspiratory Antero-lateral Retraction of the Inferior Zone of the Chest.*—This phenomenon is most strikingly observed in children suffering from obstructive disease of the larynx. In them the thoracic walls are yielding, while the muscular contractions are vigorous. Notwithstanding the normal predominance of diaphragmatic breathing in the child, thoracic expansion is forcibly made, and the little air that struggles through the obstruction is spent in rendering this movement possible. The diaphragm contracts no less violently than the muscles of the thoracic walls ; but owing to the expansion of the latter withdrawing all

* Stokes, *On the Diseases of the Chest*, p. 187.

the air that is available, it is unable to inflate the lower portions of the lungs, while its own descent is interfered with, and in corresponding degree it must act from the centre upon its peripheral attachments.*

We have already described inspiratory antero-lateral retraction of the inferior zone of the thorax as a feature of the abnormity of respiratory movement due to emphysema and bronchitis. The conditions present in this case and in laryngeal obstruction are no doubt widely different, but they agree in this, that the lower portions of the lungs cannot be inflated at the same time as the upper, and in both the descent of the diaphragm may be interfered with, notwithstanding the vigorous action of that muscle.†

This retraction is always an indication that the lower portions of the lungs are not being expanded, and it is met with under a variety of circumstances where this condition is fulfilled, but apart from emphysema and laryngeal obstruction most commonly in bronchitis of the graver form when the small tubes are affected.

II.—Unilateral Abnormity of Movement.

(*a*) *Unilateral Diminution of Movement.*—Pleuritic effusion is perhaps the disease which best exemplifies unilateral diminution

* "In forced inspiration, and especially when there is any obstruction to the entrance of air into the lungs, the lower ribs may be so much drawn in by the contraction of the diaphragm, that the girth of the trunk at this point is obviously diminished."—*Foster's Physiology*, p. 260.

† Speaking of a patient suffering from emphysema, Dr. Sibson remarked as follows :—"When first examined he suffered from a severe attack of bronchitis; the diaphragmatic efforts were very laborious, but the abdominal movement was only half the healthy amount, being ·12 to ·18 inch. When observed a second time after the disappearance of bronchitis, the abdominal movement was ·35 inch, while the diaphragmatic effort was inconsiderable. This diminution of abdominal protrusion, with manifestly increased diaphragmatic effort, allies in this respect emphysema and bronchitis with cases of extreme laryngeal obstruction, in which the same phenomena present themselves."—*Med. Chir. Trans.*, p. 407, vol. xxxi. In this case the diaphragm was unable evidently to accomplish its descent. This is exceptional, and the occurrence perhaps may be explained by the mobility of the chest, the patient being only 13 years old, while emphysema is most common in people of middle or advanced age.

of movement. Elevation may, however, take place to a degree calculated to deceive the eye, but it is usually accomplished posteriorly in time to the movement of the healthy side. Expansion movement is markedly deficient, although the intercostal spaces tend to recede during inspiration. In the absorption period of the disease, deficiency of movement remains until the lung has returned to its normal condition, but dense adhesions may render the abnormity permanent. Again, in the early stage of pleurisy, before the occurrence of effusion, and in other painful affections, the movement of the affected side may be restrained by nervous reflex influence. Among other causes of unilateral diminution of movement may be mentioned fibroid phthisis, pressure on a main bronchus by tumour or aneurism, pneumonia, cancer, or other growth infiltrating a whole lung, and pneumothorax.

(*b*) *Unilateral Exaggeration of Movement.*—This occurs as the result of one lung having become incapacitated, and the other having to perform the function of both. Accordingly, in such conditions as we described last, it is met with on the healthy side, and the asymmetry becomes the more striking to the eye; for while the movement of one side is deficient, that of the other is in excess.

III.—Local Abnormity of Movement.

(*a*) *Local Diminution of Movement.*—When the causes of defective movement already considered act on a portion of a lung only, this abnormity is the result. For instance, in the consolidation of phthisis, and in the effusion of pleurisy, diminished movement of the affected regions, apex and base respectively, afford valuable indications of disease. Where comparison with a healthy side can be made, the importance of the sign, even though slightly developed, is enhanced; but in phthisis and hydrothorax, of course, both sides are frequently involved.

(*b*) In this place we may mention the possible occurrence of rupture of a pleuritic effusion through the pleura and inter-

costal muscles. When this happens, the subcutaneous swelling, which forms, is depressed during inspiration, as the fluid returns to the pleural sac, and comes again into relief during expiration.

(*e*) We have also to mention the rare phenomenon of pulsating Empyema, in which a pulsating area appears in the infraclavicular and mammary regions close to the sternum, the aorta being healthy. In the cases in which this has been observed the other physical signs of empyema have been pronounced, and those of cardiac or vascular disease absent.

PALPATION.

Valuable information is afforded by palpation in a variety of morbid conditions. It is besides an useful mode of ascertaining the rate of the respiratory movements. For this purpose, in a female patient, the hand is placed over the infra-clavicular region, and in the male patient over the epigastrium, in accordance with the sexual types of respiratory movement already described.

By palpation we may investigate the

(1) Movements of the Chest Wall and Epigastrium.

(2) Fremitus
- (*a*) Vocal.
- (*b*) Tussive.
- (*c*) Rhonchal.
- (*d*) Friction.

(3) Fluctuation.

(4) Communicated Cardiac or Vascular Pulsation.

(1) *Movements of the Chest-Wall and Epigastrium.*—Where the lung is in full play, the corresponding chest wall is felt by the hand to fill out and expand, while any interference with the entrance of air into the lung, in whole or part, is revealed by diminution or absence of this movement, although the eye may detect little or nothing amiss, owing to elevation being well accomplished.

In the infra-clavicular regions this sign is of great value as evidence in favour of phthisis, and especially so when there is a striking difference between the two sides. In emphysema, the hand detects the *slight* expansion *movement* which forms a feature of the disease.

Palpation over the epigastrium is of use in pulmonary diagnosis as affording an indication of the action of the diaphragm.

(2) *Fremitus*—(*a*) *Vocal Fremitus.*—When a person speaks, vibration is communicated to the chest wall and readily felt by the hand. In women and children, owing to the high pitch of their voices, the sign is seldom available, while it is observed in greatest perfection in men possessing loud and grave voices. In health, vocal fremitus is greater on the right than on the left side, and the difference is best marked in the infra-clavicular, infra-scapular, and inter-scapular regions. Over these regions abnormities in respect to vocal fremitus are peculiarly useful. Thus, vocal fremitus being increased under the left clavicle, points strongly to the existence of consolidation of the left apex. Relative diminution or absence of fremitus over the right infra-scapular region indicates probable right pleuritic effusion; but over the left infra-scapular region relative increase of vocal fremitus resulting from consolidation of the left lower lobe may simulate the same condition. In other words, if the fremitus be found stronger over the left than over the right infra-scapular region, we may be quite sure that there is something abnormal; but the condition may result from either of two causes, namely, consolidation of the left lower lobe or right pleuritic effusion. The lower border of the right lung can be readily ascertained by the abrupt cessation of fremitus below it, as soon as the liver is reached. Consolidation of the lung increases the fremitus in the great majority of cases, while effusions into the pleura diminish it. Cases of consolidation are, however, occasionally met with where the fremitus is diminished or absent. Very considerable effusion again may fail to annul it over the *right* infra-scapular region. Consolidation accompanied by diminution of fremitus is somewhat difficult of explanation; but it is probable that the tubes passing through the affected portion of lung are in in such cases plugged with secretion. Another possible explanation is, that the consolidated lung is pressed so firmly against the chest-wall as to prevent vibration, for a pneumonic lung not very unfrequently bears the impression of the ribs.* Above pleuritic

* *Diseases of the Chest*, by Dr. Stokes, p. 323.

effusion the fremitus is generally increased in the infra-clavicular region, there being over the effusion below, diminution or absence of fremitus. Separation of the surface of the lung from the chest wall, owing to gaseous accumulation in the pleura, is accompanied by diminution of fremitus, as in the case of liquid accumulations.

(*b*) *Tussive Fremitus.*—The fremitus which occurs during the act of coughing may, it has been suggested, be of use in cases of aphonia.

(*c*) *Rhonchal Fremitus.*—Rhonchi when of low pitch are frequently perceptible to the hand by the vibrations they produce in the chest-wall. Gurgling in cavities and in the trachea and large bronchi likewise occasionally produces fremitus.

(*d*) In cases of pleurisy, especially in the later stages when the inflamed surfaces come together again after the removal of the effusion, a distinct to-and-fro rubbing or "catching" is perceived on palpation, corresponding to a sound yet to be described. A similar fremitus is rarely observed in emphysema associated with a corresponding auscultatory sign (*vide* page 75).

(3) *Fluctuation.* — This sign is very seldom employed, although it is referred to by Laennec, who remarks that it is only available in cases "where the fluid effused has made its way through the intercostal muscles, and forms an abscess in the subcutaneous cellular tissue, or in the rarer case, where the intercostal spaces are bulged out by the liquid contained within the chest." What has been called "peripheric fluctuation,* consists in giving a quick sharp fillip in an intercostal space, perpendicular to the surface, when a sensation of fluctuation will be transmitted to a finger of the other hand, firmly applied to the surface in the same space, at a short distance from the point percussed." This method is chiefly applicable in hydro-pneumothorax. Simple fluctuation is that employed by surgeons in the detection of superficial collections of pus or other fluid. It also is only possible when the interspaces are bulged.

* Dr Walshe, *Diseases of the Lungs*, 4th edition, p. 28, par. 83.

(4) In rare cases of left empyema, strong pulsation has been observed at the sternal portion of the left infra-clavicular and mammary regions, the heart being pushed to the right. This rare condition is to be recognised by the presence of the signs of empyema and the absence of those of aneurism.

FREQUENCY OF THE RESPIRATORY MOVEMENTS.

In health usually from sixteen to twenty breaths are taken in the minute. It may be laid down as a general law that any interference with the respiratory process occasions a stimulation of the controlling nervous centre, which is expressed by increased frequency of the respirations. We may consider in this relation the effect of such interference according as it operates upon—

(1) The Pulmonary Vesicles;
(2) The Larynx; or
(3) The Intermediate Tubular Apparatus.

(1) Acute diseases affecting, directly or indirectly, the vesicles of a portion of lung, while there exists no impediment to the free to-and-fro movement of air in the tubes in connection with the vesicles of the unaffected parts, are found to produce the greatest amount of acceleration. A portion of the oxygenating ground normally afforded to the blood, is, as it were, more or less abruptly cut off, no obstacle presenting itself to the full availability of that which remains. In this class may be enumerated pneumonia, pleuritic effusion, and pneumothorax. When such diseases become chronic, although the physical conditions so far remain unchanged, the acceleration diminishes, and may disappear, at least when the patient is at rest. The influence of pyrexia in promoting acceleration of respiration must be taken into account in the acute diseases. In chronic diseases affecting the vesicles of a portion of the lungs, in which case the interference with the respiratory process is gradually produced, accelerated respiration is by no means a characteristic sign,

although it is usually present in some degree. Chronic phthisis furnishes an example. When the lung space is encroached upon by abdominal enlargement, with elevation of the diaphragm, the effect is to withdraw a portion of the lungs from function. Œdema is the only condition in which the vesicles of the lungs generally, may be directly interfered with in their function, and great acceleration of respiration then results, especially if the bronchial tubes remain free from secretion.

(2) We pass on now to consider interference with the respiratory process acting at the other extremity of the bronchial apparatus, the larynx. Obstruction here must of necessity impede the tidal movement of the whole of the intra-pulmonary air, and ought, in accordance with the general law above indicated, to be a potent agent in accelerating respiration. Clinically we find that it is not so, and for a very simple reason, namely, that each respiratory act is a laboured struggle—often a double struggle—inspiratory and expiratory—and must be of considerable duration, thus negativing increased frequency of respiration, and often actually diminishing the normal frequency.

(3) The effect of obstruction situated in the respiratory tract lying between the larynx and vesicular tissue on the frequency of respiration remains to be considered. Firstly, with regard to the trachea and main bronchi. Here the obstruction is usually the result of aneurism or other tumour compressing the tubes from without, and the effect on the frequency of respiration is allied to that of laryngeal stenosis. Bronchitis in the large tubes interferes little, if at all, with the tidal movement of the intra-pulmonary air, although the calibre of the tubes may be diminished, and secretion have, from time to time, to be displaced. There is, then, no necessity for accelerated respiration in this case. When, however, the smaller tubes become involved in the disease, and not only diminished in calibre by swelling of the mucous membrane, but loaded with secretion, the respiratory process is seriously embarrassed, for bronchitis is a disease distributed usually throughout both lungs. The

acceleration of respiration called forth by it cannot, however, be compared with that commonly observed in pneumonia, pleuritic effusion, and those affections which cut off the function of a portion of lung only, leaving the tubes in connection with the active parts free from impediment. The explanation has been already indicated. In bronchitis, which, too, is so often accompanied by emphysema, each respiratory act is *laboured*, and therefore necessarily of considerable duration. In such cases there are two contending agencies at work, as regards the frequency of respiration. One is the stimulation of the respiratory centre in the medulla oblongata, tending to produce acceleration, the other is the mechanical difficulty in the accomplishment of each single respiration. Clinically, sometimes the one and sometimes the other obtains the mastery, but usually respiration is only moderately accelerated. In laryngeal obstruction we have seen that the influence of the mechanical difficulty is paramount, while in affections of the vesicular structure alone, it is wholly absent, and acceleration of respiration forms in them a characteristic sign, except when they are chronic, and even then, if a large area of lung be disabled.

We must recollect that the frequency of respiration may be greatly increased, quite irrespectively of pulmonary disease, by cardiac disorders, by certain morbid states of the blood, as anæmia and uræmia, and by diseases of the nervous system, especially hysteria. Diminished frequency of respiration is practically never a sign of disease of the respiratory organs, if we except extreme obstruction in the larynx or trachea. Irregularities in the rhythm of respiration, such as the Stokes-Cheyne phenomenon and allied disturbances, inasmuch as they are not directly connected with pulmonary disease, cannot be included among its physical signs.

PERCUSSION.

The great fact upon which this method of physical diagnosis rests is, of course, that the lungs are air-containing. When the multitudes of pulmonary vesicles which normally contain air are entirely filled with exudation, the special sound audible on percussion over healthy lung ceases, and the consolidated organ yields a sound practically indistinguishable from that afforded under similar circumstances by the liver, spleen, heart, or muscles. But between the absolute tonelessness of percussion of these solid organs and the normal pulmonary resonance there are many gradations. Four considerations call for attention in the clinical investigation of pulmonary percussion, viz., (1) the presence and volume of air; (2) the arrangement of the air in relation with the structure enclosing it; (3) the tension of the chest-wall and air-containing tissue; (4) the structure of the chest-wall. With regard to the last two points it is necessary to remember that the sound we elicit over the chest-wall corresponding to the pulmonary surface, does not depend solely on the condition of the lung underlying it, but that the osseous and cartilaginous framework of the chest materially modifies the proper lung sound. While the soft tissues are practically toneless on percussion, bone possesses a certain degree of resonance. This fact is well exemplified by percussion over the sternum, which yields a very decided resonance of its own. Again, the more yielding and resilient the chest-wall, the less does it interfere with the pure lung sound; while, on the other hand, the more resistant and inelastic it is, so much the less are we able to judge of the condition of the lung beneath, for in this case morbid processes tending

towards a diminution of intra-pulmonary air, act with much greater effect in diminishing and finally annulling resonance. Moreover, the degree to which the thoracic framework is loaded with soft tissues has an important influence on the percussion sound.

If the student percuss over a healthy chest, say in the infra-clavicular or axillary region between and over the ribs, the osseous influence in the latter case will be apparent, and he will perceive the importance of comparing the percussion of rib with rib and interspace with interspace, when a slight difference in the resonance of the two sides is suspected. When percussion is made over the clavicles and first bone of the sternum, the predominance of the osseous sound is recognisable, although even here the pulmonary and osseous sounds are intimately blended, and percussion over the clavicles is of much value in phthisis, as affording an indication of the condition of the lung. Th student must endeavour on lung-healthy patients to become acquainted with the modifications which exist in the percussion sound over different portions of the chest in persons whose lungs are healthy, for in this way only can he hope to recognise departure from the normal condition. With regard to these modifications, we would call attention to the importance of comparing together similar areas on the two sides, bearing in mind of course the anatomical differences which exist between them; for instance, that the liver somewhat diminishes the resonance of the base of the right lung; that the heart approaches the surface in the small triangular area known as the "superficial cardiac dulness," and extends its influence by lessening the fulness of the resonance of the superjacent lung; and that the drum-like tympanitic resonance of a distended stomach may be conveyed over a large portion of the left pulmonary area by "horizontal conduction." It is an important fact that there exists in most healthy chests a slight difference in the resonance below the clavicles on the two sides, the right being somewhat less resonant than the left. This normal difference, though never great, lends additional importance to an otherwise trifling

deficiency of resonance on the left side, while it impairs the value of a fairly marked diminution of tone on the right side.

By practice only it is possible to become familiar with the individual peculiarities of chest resonance met with in healthy subjects, but a careful comparison of the two sides, never to be neglected by the most experienced observer and indispensable to the beginner, will generally prevent peculiarities of the kind being taken as evidence of disease.

Tympanitic and Non-Tympanitic Resonance.—The normal pulmonary resonance is *sui generis*, and is to be heard, not described. We take for granted that the absolute tonelessness of percussion over the solid soft tissues—as the muscles of the thigh—is already known, and pass on to the consideration of a great type of percussion resonance, namely, the tympanitic. This type is exemplified in the drum-like sound yielded on percussion by the stomach moderately distended with air or the gaseous products of digestion. The difference between it and the healthy lung sound is easily appreciable to any one who has heard both. *The pulmonary resonance is not tympanitic;* it has been described as a "muffled" tympanitic sound. Under certain morbid conditions to be mentioned later, the "muffling" is lost, and tympanitic quality is acquired. The healthy lung *removed from the body* always yields a tympanitic resonance on percussion, and only when it is forcibly inflated does this quality disappear, the sound again resembling that of the lung during life or before the opening of the chest-wall. We have taken the stomach sound as typical of tympanitic resonance, but it is a variety very seldom met with over the lung.

"*Fulness*" *and* "*Emptiness.*"—From the stomach sound, to which no one objects to apply the term "tympanitic," a series of gradations can be traced without interruption, and differing only in "fulness," or, in other words, according to the volume of air thrown into vibration. Let the cheeks be percussed while the mouth is fully distended, a form of tympanitic resonance called by Dr. Walshe "amphoric" is elicited. The cavity of the mouth is not large enough for the contained air to

yield the stomach sound. Again, let the mouth be opened and the cheeks allowed to fall flaccid. On percussion of the cheeks now we shall hear the "tubular" note of Dr. Walshe and other English authors. It is much more "empty" or less "full" than the amphoric note, and fairly represents the sound audible on filliping the trachea (hence the name), and on percussion over portions of the lung under morbid conditions, to be afterwards mentioned. All the sounds named above are equally possessed of tympanitic quality, and differ only in relative "fulness."

While thus dwelling upon the tympanitic quality, we earnestly wish to show that we are urging no great departure from the established teaching of the English school. Dr. Walshe, describing morbid varieties of quality, mentions the following:—*

1. Hollow { Tubular / Amphoric } *Tympanitic.*
2. Tympanitic

We include all three under the one name "tympanitic" (as indicated by the bracket to the right), distinguishing them only by their varying "fulness," although necessarily other peculiarities, as changes of pitch, resistance, duration, &c., accompany each variety. It may be said that in so doing we lose in definition; but do these sounds not actually run insensibly the one into the other? To quote from Dr. Walshe himself,† "The distinction of the amphoric and tympanitic qualities cannot be set aside practically, for they are absolutely different to the ear, *yet they are allied in mechanism.* In the abdomen, for instance, a knot of intestine distended to the full with flatus will yield a sound of tympanitic quality; if the distinction becomes less by a movement onward of some of the flatus, the sound will often instantly become amphoric; this holds true whether there be notable fluid or not in the spot."

* *Diseases of the Lungs*, p. 69, par. 205.
† *Diseases of the Lungs*, p. 73, par. 215.

Again, "but in point of fact the stomach and colon *are rarely distended enough* for the conversion of their common amphoric into tympanitic quality." Paragraph 218 of the same classic work begins thus:—"On the other hand, tubular *or* amphoric quality is not very uncommon over parts acutely hepatized."

Quoting Skoda, Dr. Walshe says,*—"'If the lung contain less than its normal quantity of air, it yields a sound which either approaches to or is distinctly tympanitic.' Skoda is unquestionably right as regards the matter of fact, provided the word be understood to mean tubular or amphoric, and the reduction of the air be limited in amount." It is complained that Skoda did not define the tympanitic quality; but no description in words can give an adequate idea of the peculiarity of the sound implied. All that can be done is to refer to easily obtainable examples, and request the student to listen to them himself. Skoda adopted this method, and no one who reads his work can remain in doubt as to what he meant by the tympanitic quality. We have said enough to indicate the meaning of Skoda's classification of percussion sounds into "tympanitic" and "non-tympanitic," and into "full" and "empty" sounds.

Pitch.—The very different importance attached by Skoda† and Walshe respectively to the pitch of percussion sound is worthy of remark. Thus Skoda wrote,—"Variations in pitch are most readily detected in sounds which are clear; but they are of little value in practice." On the other hand, Dr. Walshe‡ remarks, "as a single property of percussion sound, clinically considered, emphatic alike in its diagnostic and prognostic signification, I have no hesitation of placing pitch in the first rank." As diminution of air takes place in a lung, the pitch rises; hence pitch is usually raised when the pulmonary resonance becomes tympanitic (amphoric§ or tubular) from commencing hepatization. The pitch of pulmonary percussion is

* *Diseases of the Lungs*, p. 74, par. 220.

† *Markham's Translation*, p. 19.

‡ *Diseases of the Lungs*, p. 48, par. 142.

§ We have heard a percussion sound exactly resembling that of the stomach over the right lung affected with pneumonia, but this is rare.

influenced by the tension of the chest-wall; the greater this is, the higher the pitch. If a full inspiration be made, and the breath held while percussion is effected under the clavicle, a rise in pitch is perceptible, although, with the exception of the tension of the chest-wall, all the other conditions present would tend to lower the pitch. Changes in pitch are usually accompanied by alterations in the other properties of the sound.

Over the tympanitic resonance of a cavity there is one important sign in connection with pitch, provided there be free communication between the cavity and the bronchi, namely, that it is higher when the mouth is open, lower when the mouth is shut, lower still when the nostrils are also closed.

"*Clearness*" *and* "*Dulness.*"—The properties of percussion sound to be considered next are those of "clearness" and "dulness," which must be understood, like "fulness" and "emptiness," as relative terms only. When the chest of a stout patient is percussed, difficulty is experienced in bringing out the normal resonance, though the lungs are perfectly healthy; the thick layer of soft tissues dulls the sound of the resonant lung beneath them. On the other hand, when the emaciated chest-wall of a patient dying of abdominal cancer is percussed, we remark the facility with which pulmonary resonance is elicited, provided the lungs be healthy; the thin wall of the thorax in the latter case opposes little or no obstacle to the resonant vibrations of the lung beneath. If we cover experimentally a lung on the post-mortem table with layers of liver substance, as we go on increasing the thickness of liver, we dull or deaden the pulmonary resonance until it ceases to be audible. In the case of pleuritic effusion, we have a double factor at work in the production of dulness, for not only is there the intervening layer of non-resonant fluid, but the air is necessarily squeezed out of the lung by the accumulation.

To illustrate "clearness," we borrow an example from clinical medicine, and take the case of a pneumonia spreading from the deep parts towards the surface. While yet a thin layer of air-containing lung remains, a perfectly clear, but very empty, per-

cussion sound of tympanitic quality is heard until the hepatization reaches the surface, when resonance ceases.

In two ways, then, may the normal lung resonance be replaced by the thigh or absolutely dull percussion sound:—(1) By the interposition of non-resonant material between the chest-wall and lung; or (2) by a gradual diminution of the volume of air present, as in the case of lung in process of consolidation. In the latter, the normal non-tympanitic resonance will become tympanitic and gradually emptier, until perfect dulness or tonelessness is reached. "A completely dull and a completely empty sound have naturally the same significance, and they are represented by the thigh percussion sound. As a sound becomes duller, it at the same time becomes emptier. A less full sound, however, is not necessarily a dull sound; a sound may be very empty, and yet perfectly clear." *

We have now described the four properties of percussion sound made use of in Skoda's classification, which is as follows:—

1. Full and empty.
2. Clear and dull.
3. Tympanitic and non-tympanitic.
4. High and low, with reference to pitch.

We believe that the above classification of percussion sounds furnishes the clinical observer with all that he requires for describing the sounds elicited by percussion of the chest in disease. The resistance *felt* is no doubt of much importance, and we shall speak of it afterwards.

It will be our endeavour, now, to reconcile the teaching of Skoda with that of the English school, of which Dr. Walshe may be regarded as the exponent. According to the latter author, of every percussion sound four properties have to engage the attention of the listener:—1. *intensity or mass of tone;* 2. *pitch;* 3. *quality;* and 4. *duration.*

1. *Intensity or Mass of Tone.*—This corresponds closely to Skoda's "full" and "empty." "The viscera are indeed,

* Skoda, *Markham's Translation*, p. 112.

practically speaking, almost non-sonorous in themselves . . . . . Hence the resonance of the lungs, of which we speak clinically, depends not on their proper tissue, but on the air they contain, and on the construction of the case in which they are contained." * The intensity or mass of tone, as well as the "fulness" of percussion sound, will then vary with the volume of air underneath the part struck. The larger this is, the greater the intensity or "fulness;" the smaller it is, the less the intensity, the more the "emptiness" of the sound on percussion.

2. *Pitch.*—We have already commented upon this property of sound, and called attention to the different importance attached to it by the German and English authors respectively.

3. *Quality.*—We have already shown that the percussion sound of the chest-wall corresponding to healthy lung is wanting in tympanitic quality, while percussion of the partially collapsed lung, after removal of the chest-wall, possesses tympanitic quality. The former sound we can call by no better name than "pulmonary;" it is, as we have said, *sui generis*, and must be carefully studied over different parts of the chest in relation with the lungs. Skoda's tympanitic and non-tympanitic varieties correspond pretty closely with Dr. Walshe's alterations of quality, although the latter author introduces the rather complex ideas of "hardness" and "softness," as properties of percussion sound. As these seem to us always to correspond with increase and diminution respectively of the sensation of resistance, about which we shall afterwards have to speak, we prefer to omit their consideration in this place.

4. *Duration.*—This property of percussion sound needs no explanation. The dull sound elicited on percussing the thigh is an example of very much shortened duration, the percussion sound of a moderately distended stomach, or that yielded by the voluminous lung of an emphysematous patient (the former tympanitic, the latter usually not), are examples of increased duration, the ordinary pulmonary note of a healthy chest being taken as the standard.

* Walshe, *Diseases of the Lungs*, page 47, par. 140.

Resistance.—Percussion over an enlarged liver projecting below the ribs or other large solid abdominal tumour is accompanied by a great sense of resistance; while percussion over a stomach distended with gas gives no less decidedly a consciousness of the wall yielding to our blows, provided however that such wall be not distended to an extreme degree, which would not only increase the resistance, but diminish the resonance and shorten its duration. We have taken examples from the abdomen, where the conditions are less complex than in the thorax, owing to the influence exerted by the bony and cartilaginous framework of the latter. Pneumothorax affords the best thoracic example of diminished resistance, there being only moderate distension, while pleuritic effusion with great tension of the chest-wall offers a common illustration of increased resistance. Lung conditions which augment the amount of air within the thorax, the walls not being rendered tense thereby, diminish resistance; lung conditions which lessen the amount of air in the thorax increase resistance, and the maximum amount is reached when not only all the air is replaced by solid or fluid, but the chest-walls are distended. It seems hardly necessary to mention that the resistance is normally very much greater over the ribs than over the interspaces, and that it is especially great posteriorly and over the angles of the ribs.

Dr. Walshe, in considering the morbid varieties of qualities, enumerates them thus:—

(1) Hard, Wooden
(2) Hollow, { Tubular. / Amphoric. / Cracked jar or cracked metal.
(3) Tympanitic.*

The first quality is spoken of as "resembling that of the sound yielded by mediate percussion of a deal table, and distinctly conveying the idea of hardness." Further: "The duration

* *Diseases of the Lungs*, page 69, par. 205.

of the sound having this quality is commonly less, the pitch higher than in the natural state, and the sense of resistance experienced by the fingers unusually great." We have already expressed our opinion that nothing is gained by admitting a quality of "hardness" in the sound, apart from increased resistance *felt* on percussion.

Cracked-Pot Sound.—We have yet to consider the so-called cracked-jar or cracked-pot sound. This sound differs from the other percussion resonances we have considered, inasmuch as it owns a different and compound mechanism. An idea of its character can readily be obtained by bringing together the palms of the hands, so as to leave a small space enclosed between them, the fingers being crossed together, and then striking the back of one of the hands against the knee. It will be apparent that an important element in the sound produced, is the noise of the rush of escaping air, as the size of the small chamber between the palms is diminished by the blow. It is the same with the cracked-pot sound as elicited over the chest. In the case of a large cavity, communicating with a bronchus, the sound of the air driven out by the percussion stroke mingles with the common amphoric (tympanitic) note of such a cavity, and the combination results in the typical cracked-pot sound. Necessarily the wall of the cavity, whether formed of little more than the thickened pleura and chest-wall, or of a layer of consolidated lung structure of greater or less thickness, must be capable of yielding to the blow, and in proportion as it is so, will the degree of perfection of the sign vary. There must be no obstacle to prevent the ready exit of air; accordingly we find that closure of the mouth and nostrils interferes with the cracked-pot sound, while the amphoric quality remains. Opening the mouth is therefore conducive to, and in most cases necessary for, the production of this special sign.

Percussion, with a view to elicit cracked-pot sound, should be made during expiration, after a deep inspiration, and the fingers or hammer should, contrary to the general rule, be allowed to rest for a moment on the surface. A sound resembling closely,

if not identical with cracked-pot sound, is not unfrequently heard over a portion of chest-wall, beneath which there is healthy lung, or at least no cavity. This happens in cases where the chest-wall is unusually elastic, and no doubt the sound owns a similar mechanism to that we have described above, only the sound of the escaping air will mingle with a pulmonary instead of an amphoric note. There is ample similarity between the true and spurious cracked-pot sounds to mislead even a trained ear. Above pleuritic effusion cracked-pot sound is occasionally observable, and even in partial consolidation it may occur, but in both instances the noise produced by the rush of escaping air will mingle not with pulmonary resonance, but with a resonance possessing tympanitic quality, and thus the similarity if not identity with the cracked-pot sound of a cavity is readily explicable. It was at one time supposed that the presence of air and fluid in a cavity was necessary for the development of the sign; this is not the case, but a sound resulting from the commotion in the contents of a cavity partially filled with fluid, is sometimes heard in addition and mingling with the cracked-pot sound.

From a consideration of the causation of this peculiar sound, it may be expected that the following conditions will favour its perfect development:—an elastic chest-wall and the presence of a large cavity near the surface, surrounded by consolidated lung tissue, and communicating freely with a large bronchus. In such a case the percussion blow will bear directly upon the air within the cavity, and force out a portion of it. Clinically, the anticipation is borne out.

Having now considered the various modifications of pulmonary percussion sound met with in disease, we are in a position to enter more fully into the causes upon which these depend. The normal pulmonary resonance having, we have seen, as its source the air contained within the vesicular structure of the lung, may depart from the normal in the direction of hyper-resonance or its opposite dulness. The morbid changes which produce departure from the normal in the former direction, are

of course those which increase, in proportion to the containing structure, the volume of air percussed. The disease known as emphysema will occur to every one as affording theoretically at least, an example of hyper-resonance. We are glad to commence with this form of increased resonance, inasmuch as it is one which by no means generally possesses tympanitic quality. It is a too common statement that emphysematous lung yields, when percussed clinically, a tympanitic sound. Such resonance is the exception, not the rule. No doubt, there is a natural tendency to associate tympanitic quality with a superabundance of air in the parts percussed, and *vice versa*. The danger of this tendency cannot be more forcibly illustrated than by reminding the reader of the fact that a lung in process of consolidation generally yields a percussion sound distinctly possessing the tympanitic quality. In emphysema the percussion sound corresponds with Dr. Walshe's second type of morbid resonance.

Intensity or Mass of Tone.	Pitch.	Quality.	Duration.	Resistance.
Increased.	Lowered.	Softened more or less, approaching tympanitic.	Increased.	Decreased.

Before leaving the subject of emphysematous percussion sound, it is necessary to point out that, besides the increase of resonance over the normal lung area, there is an increase of that area itself; parts which ought to be non-resonant become resonant. Thus the border of pulmonary resonance descends; the triangle of "superficial cardiac dulness" is encroached upon, and the first bone of the sternum yields a nearly pure pulmonary note, the normal osseous character being little perceptible. There is not uncommonly met with in phthisis a misleading sign in connection with partial emphysema. Caseous nodules at the apex of the lung are frequently surrounded by emphysematous structure, so that any dulness which the nodules might give rise

to, is more than masked, and the affected apex becomes more resonant than the healthy one. Another condition increasing the thoracic resonance is the presence of air in the pleura or pneumothorax. The lung collapses, and the resonance is due to air in the pleura. As a rule, and especially when the thoracic walls are yielding, a tympanitic sound of considerable fulness is elicited on percussion. Great distension of the thoracic-wall will, however, remove the tympanitic quality, and if carried to an extreme degree annul resonance. Pneumothorax seldom exists for any time alone; fluid effusion soon occurs, and causes dulness at the lower parts of the pleura. This dulness will readily change position on varying the posture of the patient; and as a further sign of the condition, we have displacement of the mediastinum. When the left side is affected, the heart is pushed over to the right, producing abnormal dulness to the right of the sternum. When hydro-pneumothorax is developed on the right side, the liver is lowered, and the heart pushed towards the left.

In passing from the morbid alterations of thoracic resonance in the direction of extra resonance to those characterised by the opposite quality, that of tonelessness or dulness, an important intermediate group demands our attention. We refer to the changes which occur when a healthy lung is becoming consolidated, as in pneumonia. Before the resonant but non-tympanitic percussion sound of healthy lung gives place to dulness, the curious change we have already mentioned is generally developed, namely, *tympanitic quality is acquired;* the fulness of the sound diminishes notwithstanding, its pitch rises, and its duration is shortened until dulness is reached. The importance of the fact is very great. Skoda thus refers to it:—"That the lungs, partially deprived of air, should yield a tympanitic, and when the quantity of air in them is increased a non-tympanitic sound, appears opposed to the laws of physics. The fact, however, is certain."* The peculiar tympanitic resonance met with above the dulness of a pleuritic effusion, has been already mentioned.

* *Markham's Translation*, p. 13.

This is the sound which, very shallow or empty while distinctly tympanitic, has been called "tubular" by the English school. It is shallow, because the volume of air percussed is small; before the effusion has reached so high, an amphoric resonance is frequently elicited over the portion of lung still in contact with the chest-wall. The cause of the assumption of a tympanitic quality by the lung resonance in these cases is not very clear, but we may regard the condition present as allied to the semi-collapse of the normal lung after the chest-wall has been removed.*

We have now to consider those morbid percussion sounds which depend upon the displacement of the normal pulmonary air by fluid or solid. The essential condition necessary for the normal resonance of the chest is, as already stated, the air contained within the lungs; when that air is removed, the resonance depending upon it must give place to the sound elicited on percussion of the soft organs generally—the percussion sound of the thigh—somewhat modified by the bony and cartilaginous framework of the thoracic-walls. This result may be brought about in two ways,—(1) by the occurrence of exudation into the air-cells up to repletion, or (2) by the encroachment upon the lung space, of pleuritic effusion or solid growth either extra- or intra-pulmonary. It requires a thick layer of soft solid or fluid to annul pulmonary resonance; but, as already stated, when fluid accumulates in the pleura, there is a double element in the production of dulness. There is the muffling of sound produced by the intervention of a non-resonant layer of fluid, but there must have been at the same time a proportionate removal of air. What resonance may remain becomes tympanitic; it must be a shallow resonance, because the volume of air is small, and it is muffled by the intervening layer of fluid. If we add to these facts the circumstance that fluid first collects at the lowest part of the pleura, which is behind where the chest-wall offers much resistance, it might be anticipated that changes of quality in the resonance would be little perceptible below the upper level of the fluid, and that dulness or tonelessness would soon super-

* *Vide* p. 36.

vene. Absolute dulness at the lower part of the pleura then is usually the first percussion change detected in cases of pleuritic effusion. The resonance of the lung above, where it remains in contact with the chest-wall, is at first unaltered, and it is only when the effusion mounts up that the resonance begins to acquire tympanitic quality in the amphoric and tubular varieties. The dulness due to pleuritic effusion is usually higher behind than in front, at least while the fluid remains moderate in amount; the transition from resonance to dulness is also less abrupt behind than in front. The dull and clear areas, as a rule, do not undergo alteration on varying the posture of the patient. In hydro-thorax such alteration may be met with, but by no means so commonly as might be expected. In hydro-pneumothorax, however, the interchange of dulness and resonance following the displacement of the air and fluid in obedience to gravity, on the assumption of different attitudes by the patient, is perfect, if there be no adhesions. When the fluid in pleurisy becomes very great, important percussion signs are afforded by changes in the position of the viscera. Thus, occurring on the left side, the effusion pushes the heart over to the right, causing dulness to the right of the sternum of cardiac origin; moreover, its own dulness may extend beyond the middle line. When a right pleuritic effusion becomes copious, it pushes the heart to the left; but the most noticeable feature is depression of the liver, which is apt to become enlarged besides, from the embarrassed pulmonary circulation acting through the right side of the heart.

Loss of resonance, due to the presence of exudation within the vesicular structure of the lung, remains to be considered. We have already described the acquirement by the resonance of *tympanitic quality*, and the progressive emptying of the sound accompanied by rising pitch and increasing sense of resistance, which precedes the final stage of dulness. When consolidation proceeds from the surface inwards, as the superficial layer of lung becomes hepatized, it will muffle the resonance of the yet air-containing portion beneath; while, if the morbid change pro-

ceeds in the opposite direction, there may remain a thin layer of resonant lung backed by consolidation, in which case resonance, perfectly clear, of tympanitic quality, and very empty, would be preserved over the affected area. It is commonly said that the dulness over pleuritic effusion is more absolute than over hepatization, and we think rightly. The tension of the chest wall may explain this, and a hepatized lung not unfrequently contains a little air, as evidenced by its yielding a shallow tympanitic sound on percussion when removed from the body.

Dr. Walshe thus describes the type of tonelessness or dulness :—

Intensity or Mass of Tone,	Pitch,	Quality,	Duration,	Resistance,
Diminished.	Raised.	Hardened.	Lessened.	Increased.

In this description we take exception only to the quality. "Hardened" conveys to us the idea that the sound resembles that of striking something hard, such as bone ; while the percussion sound over a pleuritic effusion or a tubercular or pneumonic consolidation, in no way possesses any similarity to that over the hard tissues. We prefer to consider the *feeling* of resistance in percussion, altogether apart from alteration of the sound.

We have now traced the changes which occur in the percussion sound when the healthy lung passes into a state of consolidation, or becomes airless by compression from without. The way back, if the lung be to return to its normal condition, need not detain us, for we should have only to retrace our steps. Unhappily, the lung, in the first case, may again become air-containing in another and disastrous way, namely, by the process of breaking down and excavation. With a view to this consideration, we posited as an element to be considered in percussion resonance "the arrangement of the air in relation with the structure enclosing it."* Were a healthy lung to break down into a collection of small cavities without previously having undergone consolida-

* Page 34.

tion, but little alteration in the percussion sound would be produced by the change, and it is to the consequences of the consolidating process that we owe much of our knowledge of the diagnosis of excavation. Even were a large cavity to be formed *in the midst of healthy lung*, little or no departure from the normal percussion sound would be detectable. It is the fact that excavation is almost unknown apart from previous solidification, which enables us clinically to recognise the existence of cavities In other words, cavities, clinically and pathologically, are found surrounded by solid tissue. The cracked-pot sound has already been fully described, and the other morbid qualities given to the pulmonary percussion sound while resonance remains by the combined solidifying and excavating processes may be summed up shortly by the simple expression "acquirement of tympanitic quality." The percussion sound over cavities varies in fulness, pitch, duration, and clearness (and accompanying resistance *felt*), according to the size of the air-containing space and the thickness of the intervening solid tissue. Over cavities of considerable size, the sound, when not of cracked-pot character, is usually amphoric; over those of small size, tubular; and when the intervening layer of consolidated tissue is thick, there is dulness.

The diagnosis of a cavity or cavities, large or small, cannot, of course, be made by percussion alone, but may generally be accomplished by combining the results of this method with those of auscultation.

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AUSCULTATION.

THE BREATH SOUNDS OF HEALTH.

There are two great types of normal breath sound—the *vesicular* and the *bronchial.* The latter name is somewhat unsatisfactory, and is retained here on historical grounds alone. No harm can accrue from its use if we carefully define what we mean by it. The historical warrant for the employment of the word bronchial is contained in the following sentence from Laennec: *—"Bronchial Breath Sound. I shall describe under this name, or for shortness under that of bronchial breathing, the sound produced by inspiration and expiration in the larynx, trachea, and large bronchial trunks situated at the root of the lung." The English classic author, Dr. Walshe, uses the term "bronchial respiration" in a different and more limited sense for the breath sound "audible between the scapulæ, at the upper end of the sternum, and the sterno-clavicular angles, in the sites corresponding to the bifurcation of the trachea."† These breath sounds do indeed differ somewhat from those of the larynx and trachea, and in what respects we shall point out when we have described and defined bronchial breath sound according to our own acceptation of the term.

Vesicular Breath Sound.—Vesicular breath sound represents the respiratory movement of air within the pulmonary vesicles and adjoining passages, and is audible over every portion of the chest-wall corresponding to the surface of

* *Traité de L'Auscultation Médiate.*

† *Diseases of the Lungs*, p. 92, par. 263.

the lungs. Its characters are decided; and although there exist many individual variations, this breath sound does not, in the absence of disease, depart from the broad characteristics we are going to enumerate. To hear the sound in its purity, let the student apply his ear over the infra-scapular region of an adult, keeping one hand resting on the chest to inform him of its movements. During inspiration there will be heard a soft breezy murmur, not commencing in, but ascending to, its maximum intensity, which is barely maintained up to the end of the act or beginning of expiration.

In quiet breathing we must regard expiration as the result of the cessation of muscular contraction, allowing the elastic lung and chest-wall to resume the state from which they were removed by the contraction of the inspiratory muscles. The recoil necessarily takes place immediately on the muscles ceasing from action, and accordingly the expiratory breath sound follows the inspiratory without the intervention of a silence or pause. The whole expiratory movement, however, is not soniferous, and in quiet breathing no part of it may be so. It is the latter part of expiration which is silent in healthy vesicular breathing, and the sound is loudest at its commencement, and rapidly dies away, to cease before the movement is complete. Absence of the expiratory breath sound is practically never indicative of disease, while most morbid conditions of the lung occasion its prolongation and exaggeration. It is usually three or four times shorter, and is of lower pitch than the inspiratory sound. By careful inspection of the chest movements Dr. Walshe, estimating the whole respiratory act as 10, gave 5 as the duration of inspiration, 4 as that of expiration, leaving 1 to represent the pause between the end of expiration and the commencement of the succeeding inspiration. The exact termination of expiration was difficult to determine. It will be apparent from these considerations how different is the duration of expiration, as manifested to the eye and ear respectively by inspection and auscultation. Both determinations, however, agree in this, that expiration follows upon inspiration without

interruption. No verbal description can give an adequate idea of the vesicular or any other breath sound, and it is essential that the student should hear the sound itself and make himself familiar with it. But it is hoped that the characters described will help him, while listening to the actual sound, to form in his mind a clear conception of one of the great landmarks in pulmonary auscultation.

In children the vesicular breath sound is much louder than in adults, and expiration is better heard. When a portion of lung has become disabled, and its functions have to be performed by adjoining portions, an abnormally loud vesicular breath sound is audible over these latter parts, resembling the breathing of the child, and hence called *puerile.** It is said that in such cases expiration is relatively, though slightly, prolonged; but whenever the breath sounds are exaggerated, more of the expiratory movement becomes soniferous.

Bronchial Breath Sound.—The other great type of normal breath sound—the bronchial—presents many features contrasting strongly with those we have just described as belonging to the vesicular type. In the latter there is no pause between inspiration and expiration, and expiration has only one-third or fourth of the duration of inspiration. Moreover, in vesicular breathing the maximum intensity of the inspiratory sound is not reached at the commencement, and is barely sustained to the end; while in the case of expiration the sound becomes rapidly fainter from the beginning, so as to cease considerably before the termination of the corresponding movement. Lastly, the quality of the sound itself is soft and breezy, though subject to variation not affecting its fundamental characteristics. In all these respects the respiratory sounds of the trachea differ materially. Between the inspiratory and expiratory sounds there is a very pronounced pause or silence; both sounds are of the same duration, or nearly so, and each commences and terminates with the intensity maintained throughout its course. In place of being soft

* *Vide* page 58.

and breezy, the tracheal sounds are rough and harsh, and of higher pitch than the vesicular; they give to the ear, besides, a marked impression of dryness. Skoda endeavoured to represent all the respiratory murmurs by the pronunciation of a consonant and a vowel in association.* Although we discard this system, his description of the character of bronchial breathing is so admirable, that it seems too valuable to omit from any account of the respiratory sounds. Skoda described the bronchial quality as represented by the sounds produced by inspiring and expiring forcibly through the mouth arranged to utter the consonant "ch" guttural in combination with a vowel which varies according to the pitch of the sound we wish to imitate. By the presence of this "ch" quality we are always able to identify a respiratory sound as belonging to the bronchial type—a type of breath sound the best example of which is heard over the trachea. The varieties of morbid breath sound belonging to this type, which we have to consider in another place, may indeed be regarded as derivatives merely of tracheal breathing.

Before passing from the subject of bronchial breathing, it is necessary to take into consideration a certain imperfect variety of it, to which the name has been exclusively restricted by Dr. Walshe. We refer to the breath sounds "audible between the scapulæ at the upper end of the sternum and the sterno-clavicular angles, in the sites corresponding to the bifurcation of the trachea."† These, it will be remembered, were included with the tracheal sounds under the term "bronchial" by Laennec. At the spots named the breath sounds have lost the characteristics of the vesicular type on the one hand, while they have scarcely acquired those of the bronchial. The breath sounds referred to differ from the vesicular, inasmuch as they are harsh, of uniform intensity throughout their course, and are distinctly divided by a pause; moreover, the expiratory sound is of as long duration as the inspiratory. They differ, however,

* *Markham's Skoda*, p. 85.

† *Diseases of the Lungs*, p. 92, par. 263.

from the tracheal sounds by the "ch" quality being often indistinct or wanting, by being less intense and harsh, lower pitched, and conveying to the ear less of that impression of dryness so characteristic of tracheal breathing. In different healthy individuals the degree of approximation to the tracheal type presented by the breath sounds audible over the areas named, varies. In some cases we can at once pronounce these sounds to be perfectly developed "bronchial," that is, possessing the "ch" character and all the other attributes of tracheal breathing; in other and more numerous cases the breath sounds over the bronchi lack the "ch" character of tracheal breathing, and differ from it in the other respects mentioned above. This latter variety corresponds to the normal "bronchial respiration" of Dr. Walshe, whose words, in speaking of morbid breath sounds, we quote:—"It seems, too, that the term bronchial should be limited to morbid respiration, simulating that naturally heard in the bronchial tubes." * We have called attention to the varying degree of resemblance of the breath sounds audible over the roots of the lungs to the like sounds of the trachea, and in confirmation of our views we may quote further the following statement of Dr. Walshe:—"In some persons the respiration is very purely pulmonary, even between the scapulæ." † We regard, then, the breath sounds heard over the large bronchial trunks as only imperfect tracheal or "bronchial" breathing—imperfect, because the tubes are here more or less surrounded by air-containing lung substance, which, while impeding the conduction of the tubular sounds to the surface, still further interferes with our hearing them in their purity by a new sound-production within itself. The varying degree of approximation to tracheal breathing observed in the sounds referred to, is, we believe, explicable by the existence of anatomical variations, which in some cases leave the bronchi less covered than in others.

In conclusion of this subject, let us clearly define bronchial

* *Diseases of the Lungs*, p. 101, par. 292.
† *Diseases of the Lungs*, p. 92, par. 263.

breathing in its perfect form:—A breath sound is bronchial when it possesses the quality represented by the sound produced by breathing in and out through the mouth formed to utter the consonantal sound "ch" guttural in combination with a vowel which varies according to pitch. Such breath sound always presents the rhythm we have described as belonging to the type.

MORBID BREATH SOUNDS.

We have dwelt, it may seem tediously, upon the two great types of normal breath sound, for we believe that much of the success of the student depends upon a due appreciation of their characteristics, inasmuch as morbid breath sounds are capable of classification according as they may be regarded as modifications of one or other type. There are some morbid breath sounds which cannot be regarded as belonging to either type, and these we shall refer to, after considering the others. The vesicular breath sound we know to imply the movement of air in the vesicles and adjoining passages, of the lung itself, and where we hear such breath sound we are assured that air is entering that portion of the organ over which we listen.

In diseased conditions annulling the entrance of air into the lung vesicles, the vesicular murmur disappears, and frequently in place of it a breath sound, unmistakably a modification of normal bronchial breathing, becomes audible. This latter breath sound informs us with certainty that there exist conditions which prevent the passage of air into the air-cells and minute passages, and not less surely that the intra-pulmonary bronchi or abnormal cavities are surrounded by consolidated airless tissue. We know by experience that this is the single condition which gives origin to the presence of bronchial breath sound over portions of the chest-wall, where normally there should be vesicular respiration. To take an example in pleuritic effusion. Diminution of vesicular breath sound down to silence is the first sign afforded on auscultation. This results chiefly from compression of the vesicles lying superficially, and partly from the intervention of

the layer of fluid between the source of the sound and the chest-wall. When, however, the effusion reaches a greater degree, weak bronchial breathing becomes developed, for then the lung tissue is so compressed around the still patent bronchi, whose cartilaginous walls resist the external pressure that it ceases to contain air, and the condition which we know to be associated with the presence of a bronchial breath sound in the place of the vesicular breath sound of health, is developed. The advent of bronchial breath sound is then in such cases not a sign of diminution of effusion, but the opposite. Consolidation of the lung from exudation into the air-cells has the same effect, in greater intensity, of conducting bronchial breathing to the surface through the still permeable bronchi.

To recapitulate:—Morbid breath sounds of bronchial type heard where normally there should be vesicular breathing, afford us certain information that air has ceased to enter the vesicles of the lung, and that the organ around the larger bronchi or it may be excavations formed within it, is consolidated. But a breath sound which is neither vesicular nor bronchial gives us, *per se*, no reliable information as to the condition of the lung, and well merits the name given to it by Skoda of Indeterminate (unbestimmte).

It is probable that bronchial breathing would be audible over healthy lungs were it not lost in traversing the air-containing parenchyma, which emits its own sound. In rare cases, in the absence of consolidation or effusion, the normal breath sound of the bronchi is so intense as to struggle through the air-containing vesicular structure of the lung, and be audible during forced respiration over and above the vesicular sound. This actually happens only in the neighbourhood of the large bronchi, and a careful survey of the general features of the case will prevent any erroneous conclusion being drawn from the circumstance. The beginner in auscultation must avoid mistaking pharyngeal and oral respiratory sounds, which are sometimes heard all over the chest, for the bronchial breathing of disease. Such sounds are often audible at some distance from the

patient, and a few instructions to him will effect their cessation in most cases.

We shall consider the morbid breath sounds in three groups.

(1) Breath sounds which may be regarded as modifications of the normal vesicular type.

(2) Breath sounds which may be regarded as modifications of the normal bronchial type.

(3) Breath sounds which do not belong to either of the normal types, and are not Rhonchi, Râles, nor Friction Sounds.

GROUP I.

1. Exaggerated Breath Sound,
2. Diminished „ „
3. Jerking „ „
4. Harsh „ „
5. Prolongation of the Expiratory Sound,
6. Divided Breath Sound,

} Vesicular Type.

GROUP II.

1. Bronchial Breath Sound,
   (*a*) Tubular,
   (Diffused Blowing Respiration),
   (*b*) Cavernous Breath Sound,
   (*c*) Bronchial Respiraton of Dr. Walshe,
2. Bronchial Breath Sound, accompanied by Amphoric Echo and Metallic Tinkling,

} Bronchial Type.

GROUP III.

Indeterminate Breath Sound.

GROUP I.

Modifications of the Vesicular Type.

1. *Exaggeration of Vesicular Breath Sound.*—This has been already referred to under the term puerile, in the preceding

section, and is indeed only indirectly the expresion of disease. When a portion of a lung becomes disabled from any cause, extra work is thrown upon the parts which remain active, and over these, vesicular breath sound becomes exaggerated. Expiration is said to be relatively prolonged to a slight degree.

2. *Diminution of Vesicular Breath Sound.*—We have already mentioned pleuritic effusion as a cause of diminution of breath sound, and explained its mode of action. Diminution of breath sound may be brought about, in the same manner by pneumothorax, and by diseases which occasion obstruction to the entrance of air at the larynx, trachea, or bronchus. It occurs also in certain stages of pneumonia, in emphysema (where it is the inspiratory breath sound which especially fails), and in pleurodynia. In many cases diminution of breath sound passes into suppression of the same.

3. *Jerking Breath Sound.*—Sometimes the inspiratory sound is interrupted in its course. The same phenomenon is rarely heard with the expiratory sound, which is then prolonged. Jerking respiration may be heard over the whole of the lungs when it is a purely nervous result, and occurs in hysteria and allied conditions, or in cases where inspiration is painful, as in pleurisy and pleurodynia. The local form is of much greater importance, and is met with most frequently at the apex, as a sign of incipient phthisis. A kind of interrupted respiration has been called of "cogged wheel rhythm," but is really of the nature of rhonchus.

4. *Harsh Breath Sound.*—The term sufficiently indicates the departure from normal vesicular respiration referred to. This important alteration usually sets in, accompanying the expiratory sound only, which is prolonged.

5. *Prolongation of Expiration.*—This change may be regarded as the product of two conditions,—obstruction to the exit of air and loss of elasticity in the lung structure. We meet with it in perfection in emphysema, for here both conditions are present in combination and great intensity. Obstruction to the exit of air

is accomplished by the almost invariably present bronchitis, while loss of elasticity in the lung tissue is an essential part of the disease. Prolongation of expiration rarely remains the only departure from healthy breathing. The sound is usually harsh when prolonged. It is to be borne in mind that prolongation of the expiratory portion is a feature of all the breath sounds of bronchial type.

6. *Divided Breath Sound.*—In connection with breath sound of vesicular type, this alteration occurs almost alone in one disease, namely, emphysema. It, however, like the last morbid change, is a feature of all the breath sounds of bronchial type. In emphysema the impairment of elasticity in the lung is no doubt its cause.

The last three modifications of vesicular breath sound present a strong tendency to cause the breath sound possessing them to depart from the vesicular type, even when they exist singly. When all three occur together, the breath sound can hardly be called vesicular.*

Group II.

1. Bronchial Breath Sound.

(*a.*) *Tubular Breath Sound.*—We come now to the consideration of one of the most important respiratory sounds audible in disease. In our description of the Bronchial Type, as exemplified in tracheal breath sound, we have already enumerated its essential characters. It is, however, usually of higher pitch than the tracheal sound, and apparently more rapid in its course.

Tubular breathing gives to the ear the distinct impression of a to-and-fro movement of air through a tubular space placed in the immediate vicinity of the spot examined, and frequently a sensation, as if during inspiration the air were drawn from the ear

* In emphysema exceptionally the combination may exist, while the breathing is vesicular, but the profound alterations undergone by the vesicular structure itself, in such cases, is a sufficient explanation of this apparent exception to the fundamental rules we have laid down.

of the observer, and in expiration blown back again. This sound is heard in typical perfection over a hepatized lobe in pneumonia. What are the conditions with which, from clinical and pathological experience, we are enabled to associate its presence in such a case? Not surely with a to-and-fro movement of the air in tubes traversing the solidified lung, for there can be no expansion of the vesicular structure to which these tubes lead. To explain the sound, we must admit from the outset that it is *conducted.* In speaking of the normal breath sounds, we described in detail the characters of that formed in the vesicular structure of the lung, as well as of that formed in the large tubes which lead to it. No one can be in doubt as to which type tubular breathing belongs. We are forced then to the conclusion that tubular breath sound has its origin beyond the hepatized portion of lung. It is a difficult matter to decide at what level of the bronchial tree *production* of breath sound of bronchial type ceases; but it is certain that the laryngeal breath sound formed at the glottis, and heard almost as well over the trachea as the larynx itself, is predominant down to the entrance of the tubes into the lung. Here a new breath sound is generated, in which the other is lost. Two factors are in operation to facilitate the transmission* of bronchial breathing to the surface of a consolidated lobe. One is the cessation of the breath sound of the lung tissue around the tubes, the other is the fact that the bronchi within the affected portion of lung are now surrounded by airless tissue, and are therefore in a condition which renders them more capable of conveying onwards the breath sound of the larger tubes, with which they are continuous. It is possible even that reinforcement of the same sound may take place within them. We do not enter into the question of the relative conducting properties of solid and healthy lung; for its discussion would afford us no more valuable clinical rule. Cardiac sounds are undoubtedly transmitted with increased intensity over a

* Occasionally one portion only of the breath sound assumes bronchial characters over a consolidated portion of lung. In the majority of cases, thi is the expiratory.

pulmonary apex which has become consolidated, but the case is hardly analogous, for sounds produced in air have no place in the cardiac phenomena.

To recapitulate :—Where we hear tubular breathing we know that the lung parenchyma has ceased admitting air, and that the bronchi running through it are surrounded by solid tissue, whether it be lung structure *plus* exudation or lung structure alone, simply compressed, as by fluid in the pleura. Pneumonia and pleurisy with effusion respectively supply these conditions, and as we might *a priori* expect, the breath sound, though it is as purely bronchial in character, is usually less intense and apparently more remote in the latter than in the former. The posterior position of the compressed lung must be borne in mind.

Diffused Blowing Breath Sound.—The "diffused blowing breathing," described by Dr. Walshe and other writers, requires only to be mentioned, for in all probability it is merely an imperfect bronchial breath sound. Dr. Walshe speaks of "the fact which may," he thinks, "be frequently observed, that the well-marked tubular form signifies a more advanced degree of disease than the diffused." In many cases the perfection of tubular breathing is never reached.

(*b*) *Cavernous Breath Sound.* — The next modification of bronchial breath sound is the so-called "Cavernous Breathing." Laennec introduced the term, and his own words best describe what he meant to imply by it. "Cavernous breathing.—This respiratory sound has the same character as bronchial respiration, but the air seems to pass into a larger space than that of the bronchial ramifications." Dr. Walshe remarks,—"The ear receives the impression most distinctly of connection with a hollow space." We hesitate to accept this variety of the bronchial sound. The idea of a cavity is derived from complex sources, and it will suffice here to enumerate a few features in which the breath sounds over cavities, commonly differ from tubular breathing, while still lacking amphoric echo, the only certain sign of a cavity. The pitch is usually lower, that of

expiration especially. Comparative slowness of production is said to be another feature. "There seems to be a delay in the interior of the chest" (*Dr. Walshe*, p. 104.) We have not, we feel, made out a case for the retention of this term, and we are glad, in conclusion, to be able to quote from the last named author, who advocates its retention, the following words:— "Over certain solidifications surrounding bronchi, not necessarily even modified in calibre, there may exist at once the breathing, the rhonchus, and the vocal phenomena, commonly associated with and commonly supposed peculiar to actual excavation in the lung substance."*

(*c*) *The Bronchial Respiration of Dr. Walshe.*—We have already commented on the "Bronchial Breathing" of Dr. Walshe, and here give his own description of it. After speaking of harsh respiration, he goes on to say,—"This type insensibly passes into the higher grade, *bronchial respiration*. . . . . Both sounds are now *rough* and *hard*, and notably more *dry* than in the natural state; the sharp blowing quality is heard in inspiration as distinctly as in expiration, and in the latter to a greater degree than in respiration of the previous type. The *intensity* of both sounds appears augmented, and is even greater than in normal bronchial respiration; otherwise morbid breathing sounds of this type closely resemble the natural sounds in the larger bronchial tubes."† Dr. Walshe's description of the latter sounds is as follows:—Bronchial respiration . . . . . wants in both its divisions the perfect softness and gentle breeziness belonging to the pulmonary species; both are slightly harsher, of higher pitch, more rapidly evolved, especially the expiratory, and follow each other closely, are less accurately continuous than in pulmonary respiration."‡ We are somewhat unwilling to place this variety of breath sound in the present section, and are disposed to class it with Skoda's indeterminate breath sound. As Laennec, however, in his definition of

* *Diseases of the Lungs*, p. 105, par. 304.
† Walshe, *Diseases of the Lungs*, p. 99, par. 287.
‡ *Walshe*, p. 92, par. 263.

bronchial breathing, included the breath sound audible over the "large bronchial trunks situated at the root of the lung," we can consistently consider it in this place. It must, however, be regarded as an imperfect bronchial breath sound, and is met with over partial consolidation of the lung.

2. Breath Sound of Bronchial Type, accompanied by Amphoric Echo and Metallic Tinkling.

Amphoric Echo and Metallic Tinkling.—These phenomena constitute the only certain signs of a cavity. For their production cavities of considerable size, and not merely tubes, are necessary. Smooth solid walls, capable of reflecting sound, are probably also requisite. No verbal description can adequately represent amphoric echo and metallic tinkling, but we refer to easily obtained sounds which resemble them. Amphoric echo may be imitated by blowing into a jug or decanter, or through the narrowed lips with the cheeks expanded as much as possible. Metallic tinkling, which occurs under the same conditions, and indeed is probably the same phenomenon suddenly raised in pitch, may be imitated by applying the ear to the surface of a decanter containing a small quantity of fluid, and letting drops of water fall into the vessel, upon the surface of its contents. The presence of fluid in a cavity is certainly not necessary for the production of amphoric echo, nor indeed for that of metallic tinkling. In the case of the latter, however, it may be remarked that the breaking of bubbles has a peculiar facility in producing the phenomenon. This subject, however, belongs to a later consideration. We introduce metallic tinkling in this place because it can be produced by the respiratory sounds alone and apart from râles or voice sounds. There is only one condition which can give rise to the sounds under discussion, and that is, as previously indicated, the presence of a large air-containing solid-walled cavity. Practically, such cavities are hardly ever dry. They occur in the lung as a result of excavation, but the expanded normally potential cavity of the pleura when air has

been admitted into the sac allowing the lung to collapse, forms the largest air-containing space we meet with in the chest. The question naturally arises, is the respiratory sound accompanied by amphoric echo, the product of air passing to-and-fro through a communicating bronchus in the former case, or through the same and a fistulous opening in pneumothorax? Although we are not prepared to deny absolutely this mode of origin, at least in the case of intra-pulmonary cavities, as a rule, the breath sounds audible in the conditions mentioned are the normal bronchial breath sounds transmitted through and resounding within the air-containing space. Such breath sounds are of bronchial type *plus* a superadded character, which is our present subject, and which is acquired in their passage through the cavity, whatever its origin. To take the case of pneumothorax, there is immobility of the affected side, and no passage of air to-and-fro through the patent fistulous communication, if such exist; moreover, there is collapse of the lung, and the bronchi are in consequence surrounded by solid tissue. From these tubes, surrounded only by the compressed lung tissue, the conducted normal bronchial sounds will readily pass into the pleural cavity through the thin layer of collapsed lung, whether a fistula exist or not. In their passage through the cavity to the surface, amphoric echo is acquired. In intra-pulmonary cavities, we have so far the same conditions, but a direct communication between the bronchial tubes and air-containing chamber is generally present, and usually free; while the corresponding chest-wall is not necessarily motionless, so that some passage to-and-fro of air, at all events, may take place. Deficient movement of the chest-wall corresponding to cavities (almost invariably, they are surrounded by condensed tissue) is the rule, and it can hardly be that the loud free breath sounds, which acquire amphoric echo, are due to the actual entrance of air into and its exit from the cavity, during the respiratory movements which are locally so feeble. Very much more likely is it that they are the bronchial sounds of health (in which, as we have seen the laryngeal sound, is predominant) modified by their passage through the cavity.

Group III.

Indeterminate Breath Sound.

Skoda classed breath sounds which have neither the characters of vesicular nor bronchial breathing under the name of "indeterminate"* respiratory sounds. A breath sound possessing the three following alterations from vesicular breathing, namely, harshness, prolongation of expiration, and a pause between the inspiratory and expiratory portions, and yet lacking the "ch" quality of bronchial breathing, conveniently falls under this designation. That is to say, a breath sound which is harsh in both the inspiratory and expiratory divisions, has an expiration as long as inspiration, and is distinctly divided by a pause between the two, cannot be vesicular breathing; and inasmuch as the "ch" quality is absent, neither can it be perfect bronchial breathing. Such breath sounds do exist, and do not determine for us with certainty either that the vesicular structure of the lung is still capable of expansion, or that it has ceased to be so, and become solid around the bronchial tubes from exudation or compression. They are met with in cases where there is partial consolidation of the part of the lung examined. The following words from Dr. Markham's translation of Skoda's work on Auscultation* are well worthy of attention, and form a fitting conclusion to this part of our subject. "The more delicate and practised the ear of the observer, the more readily will he judge rightly of these transition murmurs; it is, however, much the safer plan to regard all respiratory murmurs, which are not distinct in chararacter, as indeterminate murmurs, and to draw no conclusion from them without due consideration of all the other indications obtainable by percussion, &c. With such precautions, an observer, though but moderately practised in auscultation, will rarely fail in his object."†

* *Markham's Skoda*, p. 110.

† Page 121.

ADVENTITIOUS SOUNDS HEARD ALONG WITH OR OBSCURING THE BREATH SOUNDS IN DISEASE.

The difficulty of conveying to the mind of one who has not heard for himself a correct idea of the various adventitious sounds audible on the surface of the chest in disease had been doubtless experienced by Laennec, when he wrote at the outset of his chapter on these sounds:—"Words will often fail me to express their characters, or at least it will be difficult for me to describe them in a manner sufficiently accurate to give a correct idea to him who has never heard them."

The word râle, meaning a rattling in the throat, was used by the founder of pulmonary auscultation, to express all the sounds we are now considering. But he used, in precisely similar manner, the word rhonchus, denoting a snoring sound. In the preface of Laennec's Treatise, the following curious sentence occurs, immediately following a caution against pronouncing before patients the names of stethoscopic signs:—"For the same reason, I substitute habitually for the name *râle* that of *rhonchus*, which frightens nobody,* if by inadvertence one happen to pronounce it (before patients)." Some authors have reserved the term rhonchus for the so-called "dry" sounds, and râle for moist or bubbling sounds; while others, following Laennec, have used both terms indiscriminately.

In the following classification we use the word *râle* to designate all those adventitious noises which may be resolved into sounds apparently produced by the bubbling of air through fluid, the bubbles being of various sizes and formed under a variety of circumstances. We limit the term *rhonchus*, again, to those noises which cannot be resolved into the sounds of bubbling, and which result from obstruction offered to the passage of air in the bronchi, such obstruction being commonly due to the presence of viscid secretion or local narrowing of the tube,

* Râle of course, to French-speaking people is associated with the "death rattle," the only râle known to the laity.

from projection of the mucous coat or other cause. In our first division there are included sounds which appear dry to the ear, and of which the production by bubbles has been disputed, and not without reason; while in our second division the sounds, as we have just stated, frequently owe their origin to the presence of viscid *fluid* in the air-passages. Hence we have discarded the employment of the terms "dry" and "moist," commonly applied to adventitious sounds. In our third division we include pleuritic friction, and a peculiar sound owning neither of the causes we have named.

GROUP. I.—*Râles.*

1. Crepitation or Vesicular Râle.
2. Bubbling Sounds.
   - (*a*) Simple Bubbling { Small. / Large.
   - (*b*) Bubbling with Special Characters { Consonating and / Amphoric Bubbling.

GROUP II.—*Rhonchi.*

Low and High Pitched.

GROUP III.

1. "Laennec's Dry Crepitant Râle with large Bubbles" (*Le Râle crepitant sec à grosses bulles ou craquement*).
2. Pleuritic Friction.

GROUP I.—*Râles.*

1. Crepitation or Vesicular Râle.

No term in auscultation has been more indefinitely applied than this, although its characters define it accurately. The tendency has been to apply it to the bubbling sounds we have yet to describe, and in this tendency we have perhaps

some support for including crepitation under the râles. The sound can be very easily imitated by rubbing between the finger and thumb a lock of one's hair close to the ear. It consists of a series of sharp crackles, conveying to the ear the impression of the bursting of very minute bubbles of similar size. These crackling sounds occur towards the end of inspiration, and are rapidly produced. When in great abundance, nearly the whole of the inspiratory murmur may be masked by them. Cough and expectoration do not affect the occurrence of this râle, unless it be that the former rather increases it by the full inspiration which precedes. Crepitation has been called the vesicular râle, inasmuch as its presence, like that of vesicular breath sound, informs us of the entrance of air into the pulmonary vesicles, and enables us to exclude all conditions which render this impossible. True crepitation is never heard during expiration. We have already referred to the impression of dryness afforded by the sound, and a theory, other than that which attributes it to the bursting of minute bubbles, has to be mentioned. It is held by some that this râle is produced by the sudden expansion of collapsed air cells, or, it may be, minute passages collapsed and having their surfaces glued together by viscid secretion. In support of this theory is the fact that, over even healthy lungs, and especially when the patient has been lying on the back and breathing quietly, a few crackliug sounds, exactly simulating crepitation, are often audible on first listening over the bases of the lungs. These speedily disappear on a few deep breaths being taken. This spurious râle is not confined to the bases of the lungs, though most commonly met with there. On the other hand, the theory we have adopted in our classification is supported by the well-ascertained fact that crepitation in typical perfection is not unfrequently developed in pure watery œdema of the lung. Notwithstanding its occurrence in other affections, crepitation may be regarded as *par excellence* the râle of the first stage of pneumonia. We shall always be right in attributing it to abnormal conditions of the vesicular structure of the lung.

2. Bubbling Sounds.

(*a*) *Simple Bubbling.*—There is, of course, no hard and fast line between small and large bubbling sounds, the smallest large râle corresponding with the largest small râle. For purposes of description, however, it is convenient to make use of the terms.

Small Bubbling.—We have admitted that the mechanism of the last described râle is open to question, but no one doubts the bubbling character of that we are now considering. The sounds are indicative of small-sized bubbles, although distinctly larger than those we suppose to make up crepitation; moreover, they are dissimilar in size and vary in number according to the amount of fluid present. They attend as a rule both respiratory movements, but are commonly louder during inspiration. Sometimes they are limited to inspiration, and then are usually of the smallest variety. Even then this râle is distinguishable from the rapid burst of crackling sounds characterising true crepitation. Small bubbling is the special sign of capillary bronchitis, denoting the presence of fluid secretion in the minute tubes. It occurs in other diseases accompanied by the like condition. We have stated that the individual bubbles vary in size, and it is probable that they are formed in tubes of different calibre. We cannot, however, judge accurately of the magnitude of the space in which bubbles are formed, by their estimated size, inasmuch as, although large bubbles are necessarily formed in tubes or cavities of some capacity, comparatively small bubbles may be formed in large tubes or cavities.

Large Bubbling.—We have described small bubbling as varying in regard to the size of the single bubbles, from a sound resembling crepitation up to one which approaches our present subject. In treating of large bubbling we shall commence with the variety furthest removed from that last described. Tracheal râle is the sound so well known to occur in the dying. It is to this that the word râle is popularly applied in French, and hence Laennec's caution, before quoted, to avoid the use of the word in the presence of the patient or his friends. For us, of

course, there can be no such objection to the term. The bubbles are of large size and numerous, and differ from other bubbling sounds in the pulmonary passages by being audible at a distance from the patient. This râle is produced by the passage of air through abundant fluid effusion in the trachea and bronchi, which can no longer be expelled by cough. Accordingly, the sign occurs in the last stage of most diseases ; but it is specially marked where death takes place by coma, as in apoplexy and fever, or from pulmonary affections, as phthisis, pneumonia, &c. Large bubbling of this kind occurs also in pulmonary hæmorrhage, blood taking the place of secretion. When similar râles are audible over a limited portion of a lung and near the ear, their formation can only take place in a cavity (gurgling).

Fluid poured into the medium-sized bronchi, whatever the cause, of course also gives rise to bubbling. The bubbles are in this case unequal in size, though the largest is always much smaller than those of the tracheal râle, and the râle is inaudible except when the ear is applied mediately or immediately to the patient's chest. This râle occurs during both portions of the respiratory murmur, and is modified by the acts of coughing and expectorating. It varies much in the number of its bubbles, according to the amount of fluid present in the tubes. Such a râle is the special sign of ordinary bronchitis during the stage of free secretion, just as the smaller variety of simple bubbling is the special sign of capillary bronchitis in the same stage.* As the gravity of bronchitis varies in most cases in an inverse ratio with the calibre of the affected tubes, or, in other words, according to the degree of approximation to the lung substance, the so-called mucous râle is of better prognosis than the small bubbling of the capillary affection. Paradoxically, it is the largest bubbling which constitutes the "râle de la mort." This is, however, only an apparent exception to the rule. A common cold is a catarrhal affection of the respiratory tract not

* The large varieties of bronchitic bubbling sounds are frequently termed "mucous" or "submucous" râles, while the small variety is termed "subcrepitant."

usually passing downwards beyond the trachea. Secretion is no doubt thrown copiously out into the trachea, but it is never allowed to accumulate, and tracheal rale consequently does not occur. Tracheal râle derives its terrible significance, not indeed as a sign of the implication of the mucous lining of the trachea in inflammation, but because it bears unmistakable witness to the failing of the powers of life, being the necessary accompaniment of *accumulation* of fluid in the large air-tubes, while the respiratory centre is yet able to accomplish that ebb and flow of intra-pulmonary air which results from the movements of the chest-walls and diaphragm.

(*b*) *Bubbling with Special Characters—Consonating Bubbling.*—We use the term "consonating" not without regret, as it implies a theory that may or may not be true. There can be no doubt, however, as to the difference which exists between the characters of râles heard over solid and air-containing lung respectively. In the former the sounds acquire a clearness and resonance which marks them at once to the practised ear as conveyed through, if not formed in a tube or moderately-sized cavity *surrounded by solid tissue.* Skoda, who first called attention to the special characters of this morbid sound, thus describes it:—"This râle is clear and high, is formed by unequal bubbles, and accompanied by resonance which has neither an amphoric nor a metallic character." Dr Walshe, commenting upon Skoda's description, remarks,—"Under the title of 'consonating,' Skoda describes a rhonchus clear, high pitched, and unequal bubbled, accompanied with resonance which is, however, neither metallic nor amphoric. . . . . This description would answer to bubbling rhonchus produced in tubes surrounded by solid tissue, and intensified, as possibly may be the case occasionally, by unison resonance." * As the vesicular râle determined for us the entrance of air into the pulmonary vesicles, so this râle determines for us the opposite condition and the presence of consolidation round the tubes, and has precisely the same significance as bronchial

* *Diseases of the Lungs*, p. 115, par. 342.

breathing heard over the lung: it attends consolidating processes.*

Amphoric Bubbling.—Rales may acquire a further resonance which we call "amphoric," and accompanying this there is frequently another phenomenon, which we have already referred to as "metallic tinkling." Such rales are usually formed of large bubbles, but are not necessarily produced in the air-containing chamber, from which they derive their resonance, nor indeed in a large bronchial tube in immediate communication with it. Bubbles formed in a neighbouring bronchial tube of considerable size may acquire amphoric character in an adjoining pulmonary or pleural cavity. In the latter case, that of pneumothorax, a free communication between the pleura and bronchi is the exception. Skoda remarks,—"The râles can take this metallic character, although no communication exists between the pneumothorax and the bronchial tubes."† The bubbling sounds themselves, then, may be generated in the bronchial tubes of the lung, but in order that they may acquire amphoric resonance they must pass into a large air-containing chamber possessing walls capable of reflecting sound. We have already called attention to the fact that râles more readily acquire amphoric resonance than the voice or breath sounds. Metallic tinkling is best heard in cases of hydro-pneumothorax after succussion.

Group II.—*Rhonchi.*

These sounds are commonly due to the presence of viscid fluid in the bronchial tubes, hence it would seem objectionable to call them "dry" sounds. The impression of production by bubbling is, however, entirely wanting. Rhonchi are in the immense majority of cases the consequence of bronchitis or catarrh of the bronchial mucous membrane, an

* Under "consonating" bubbling sounds we would include the sounds commonly spoken of as "dry and moist crackling." These are the sounds so frequently heard over phthisical apices.

† *Markham's Translation*, p. 136.

affection usually distributed throughout both lungs, and they come to be associated with that disease, and called in consequence "bronchitic sounds." When free secretion has taken place bubbling sounds are of course heard. Rhonchi give no information as to the state of the lung tissue, for in cases where a portion of lung is consolidated, and the ebb and flow of air within its tubes has ceased, the rhonchi of neighbouring portions of still active lung may be well conducted to its surface. It is difficult to appreciate in these sounds a quality analogous to that we have termed "consonating" in the case of bubbles.

We divide rhonchi simply according to their pitch, and speak of them as high and low. An approximate inference as to the calibre of the affected tube may be drawn from the pitch. The lower or graver this is, the larger the tube in which it is formed; the higher the pitch, the smaller the dimension of the tube concerned. Various well-known sounds have been called into requisition to afford an idea of rhonchi, such as snoring, cooing, &c. Even in the same case they vary indefinitely in character from time to time, and are frequently accompanied by bubbling in greater or less abundance, as free secretion is established.

Rhonchi are usually of considerable duration, and occur with the inspiratory or expiratory, or with both breath sounds, which they quite obscure. Expiration is in any case prolonged. They are often loud enough to be audible at some distance from the patient, and under Palpation we have called attention to the tactile fremitus they may occasion. As to the cause of rhonchi, it is probably an obstruction offered to the passage of air by the presence of viscid mucus adhering to the bronchial surface, or it may be the presence of folds of swollen mucous membrane, or local projection the result of pressure from without. One point in favour of the first explanation is the fact that coughing always modifies and often removes them, so that a permanent cause is somewhat difficult to understand. Rhonchi occur in great abundance during the paroxysm of spasmodic asthma, a disease supposed to consist of spasm of the muscular fibres of the bronchi. A paroxysm is well known to be

frequently followed by free secretion into the tubes, and during the paroxysm it is difficult to eliminate a causal relationship between the rhonchi and an early viscid stage of secretion. If spasm of the tubes be considered the sole factor in the production of the sounds, in all probability the spasmodic condition is irregularly distributed, and the mucous membrane may be thus thrown into folds. Aneurism and other tumours pressing on the large bronchi are frequent causes of rhonchi, but here again it is difficult to eliminate the effects of secretion so liable to be induced under the circumstances. Enough has been said to indicate the general characters of the morbid sounds in question; they are perhaps the easiest morbid signs in pulmonary auscultation to recognise, and their very frequent occurrence gives the student abundant opportunities for making himself familiar with their many variations.

Group III.

1. "Dry Crepitant Râle with Large Bubbles." *
2. Pleuritic Friction.

The two remaining morbid signs, though differing materially in their mode of production, have yet sufficient in common to permit of our classing them together. It is interesting to note that friction sound as a sign of pleuritis and the consequent roughening of the opposed serous surfaces, escaped the attention of Laennec, while he described a friction sound as occurring in cases of emphysema, especially interlobular, in association with the "Râle crepitant sec à grosses bulles." After speaking of this râle, he goes on to say,—"One perceives commonly at the same time the sensation of one, or several bodies, which ascend and descend, rubbing along the ribs during inspiration and expiration." That emphysema alone can produce rubbing sound has been denied by writers of eminence; but Dr. Walshe gives his assent in the following words:—"I am induced to think that

* This is a very rare phenomenon, while pleuritic friction is common.

Laennec's belief respecting the occurrence of friction signs, in some forms of emphysema, was not erroneous;" and for further testimony in favour of Laennec's statement, I may refer to a case, proved by post-mortem examination, and recorded in Professor Gairdner's *Clinical Medicine*, chap. vii. p. 144.

1. *Dry Crepitant Râle with Large Bubbles.*—This paradoxical term used by Laennec sufficiently explains the reason of our removing the sound from the class of râles. Dryness and bubbles can hardly be associated together in its production. Laennec's own description shows that he considered it as produced by a mechanism other than bubbling. "It gives the sensation of air distending dry and very unequally dilated pulmonary vesicles," again, "the sound is quite analogous to that of a dry bladder being inflated." Different from the rubbing sensation referred to above, this râle, or *craquement*, is confined almost always to inspiration. It is a rare sign, and there is no doubt its frequency was greatly over-estimated by Laennec. Its probable cause is the distension of subpleural dilated vesicles which have lost their elasticity.

2. *Pleural Friction.*—We have already described the chest movements of respiration, and that there exists a certain amount of to-and-fro movement between the surface of the lung, and corresponding inner surface of the chest-wall,—in other words, between the pulmonary and costal pleural surfaces,—follows as a necessary consequence. The other factor in the production of friction noise, is the existence of a roughened condition of the two opposing surfaces. As in quiet breathing, the inspiratory movement is performed with greater force than the expiratory, which is simply a recoil, the first production of friction sound is likely to be present only during the former. Friction soon, however, accompanies both respiratory acts; in very rare cases, it may be temporarily limited to expiration.

Friction is usually best heard over the lower thoracic zone; sometimes it is limited in area, at other times widely distributed over the surface of the lung, but is rarely met with on both sides

at the same time. Where effusion separates the pulmonary and costal pleural layers, friction sound is necessarily annulled. It is common to hear friction at the side towards the front, while the silence of effusion exists behind. As the fluid becomes absorbed, the pleural layers again come into contact, and it is then that the loudest and most typical friction is heard, for the false membranes have gained much in consistency. Many different kinds of friction noise have been described, but we question how far verbal description, in this regard, is practically useful. Friction sound gives to the ear a distinct impression of its mode of production; it is superficial, and has always something of a rubbing character, varying from a faint graze to a coarse grating perceptible to the hand as well as to the ear. The sound is seldom uniform when of any duration, and frequently consists of a series of abrupt sounds produced in rapid succession. It is this character which causes friction at times closely to resemble intra-pulmonary râles. When best developed the breath sounds are quite obscured, and the friction sound exists through the inspiratory and expiratory acts, without the interruption of a pause. Its duration varies much, and it may be audible during only a small portion of either movement. In health the gliding movements of the pleural surfaces upon one another are not productive of sound, and it is only when these surfaces, as already stated, become roughened by inflammation that friction becomes audible. Even projections on one or other layer will not produce it, unless their surfaces be inflamed.

MODIFICATIONS OF THE VOICE AS HEARD OVER THE THORAX IN PULMONARY DISEASE.

Since the days of Laennec, the modifications brought about in the thoracic voice by the existence of diseased conditions in the lungs have occupied an amount of attention they hardly deserve. Dr. Walshe refers to the value of these sounds in the

following terms: *—"The signs derived from modified vocal resonance are uncertain in character and obscure in theory, and though occasionally not devoid of clinical signification, hold, as a rule, a very low place among physical aids to diagnosis." We earnestly endorse this statement. It must be remembered, however, that vocal resonance was the ground chosen by Skoda on which to do battle for his celebrated theory of consonance in the chest. Skoda's theory may be refuted, but no experienced clinical observer, who has availed himself of the physical signs of pulmonary disease, can fail to admire the remarkable skill with which the actual facts of observation are presented by him, or to appreciate the marvellous accuracy of the inferences he drew from them, as to lung conditions.

In considering the voice sounds in relation to pulmonary auscultation we have to take into account the two elements of which they are composed,—(1) The sound produced at the glottis by the vibrations of the vocal cords; (2) Articulation formed in the mouth. The articulated voice being produced in the mouth, in its passage through the trachea to the thoracic walls, has to pass through the glottis, where the approximated vibrating cords may be supposed to offer a formidable obstacle to the transmission downwards of the words spoken. We shall not be surprised then to learn that normally the articulation of the voice is rarely audible below the trachea, and that when the words can be identified at all over the roots of the lungs, they are only recognisable with difficulty. We are also prepared by the above considerations to find that in disease it is often whispered articulation which is best conveyed downwards to the chest-walls, inasmuch as the vibrations of the cords and closure of the glottis will, in the case of the whispered voice, not interfere with the transmission of words formed out of the mere expiratory murmur.

In accordance with our usual custom, we shall first describe the natural phenomena of the voice as audible over (1) the larynx and trachea; (2) the large bronchi; and over (3) the

* *Diseases of the Lungs*, p. 127, par. 400.

lung substance. It is important that the student commencing auscultation should familiarise himself with the voice heard over these localities in different individuals possessing healthy lungs. For in this way only he can establish in his mind a standard with which to compare the modifications of disease. There are many features of analogy between the voice and breath sounds, and in each of the localities named above there will be heard for the voice, as for the breath sounds, a peculiar type. Again, as we regarded the bronchial breathing of the roots of the lungs but as an imperfect representation of the tracheal similar sounds, so we may consider the voice, audible over the roots of the lungs, as only the tracheal carried down in the column of air, but obscured and rendered imperfect from local conditions. Accordingly, we speak of *bronchophony* only, including the perfect and imperfect varieties. In the case of the breath sounds, we were unable to determine where the *production* of breath sound of bronchial character ceased, although admitting the predominance of the laryngeal sounds down to the bronchial trunks at the roots of the lungs; the voice is altogether a conducted sound from the larynx and mouth. But further, we shall find that as some of the most important modifications of the breath sounds resulting from disease consisted of a replacement of vesicular breathing by bronchial, so also with regard to the voice sounds, a replacement of the vocal sound (voice we cannot call it) naturally audible over the pulmonary substance by a bronchial voice, in greater or less perfection, is the most important change we have to consider.

The Normal Vocal Sounds as heard over—

(1) The Larynx, Trachea, and Bronchi (so called Laryngophony, Tracheophony, and Bronchophony).

(2) Over the Pulmonary Substance.

(1) When we listen with the stethoscope over the trachea of a healthy adult, and ask him to speak, the voice is heard with an intensity painful to the ear. Notwithstanding the loudness of the sound, its articulation is imperfect, although when the free

ear is closed the words can usually be recognised, as is also the case during whispering; over the larynx itself the sound is of even greater intensity and the concussion to the ear more forcible.

Again, on listening over the first bone of the sternum, or behind between the scapular spines, the voice sound will be found to have diminished considerably in intensity, and it will be rarely possible to identify the words spoken when the free ear is closed. The voice here possesses to some extent the quality of that from a speaking trumpet. The degree to which the voice sound in this locality approaches the tracheal varies in different individuals, just as is the case with the breath sounds. It follows that care must be taken in drawing inferences from exaggerated intensity and clearness of the voice in the neighbourhood of the large tubes.

We propose to use simply the term *bronchophony* for morbid transmission of a voice sound similar to those we have just described, when such is heard over portions of the chest-wall which correspond to the surface of the lung. When this morbid change is most perfectly developed the voice resembles the healthy tracheal voice; when developed to a less degree, it simulates the voice as heard over the large bronchi. They are the same in essence.

The question of intra-thoracic reinforcement of sound was avoided in describing the breath sounds, although probably it holds with an equal amount of truth to them. In the case of the vocal resonance, the seat of origin of the voice cannot be questioned, for it is produced in the larynx and mouth (the articulation in the latter). When the voice is heard as loudly and clearly over a portion of chest-wall corresponding anatomically to lung substance, as over the trachea, it must have become strengthened in the chest. A satisfactory explanation of this reinforcement has not been given, but fortunately we know much of the conditions which render it possible, and these will be afterwards described.

(2) *Vocal Resonance over Healthy Lung.*—We have now to consider the vocal resonance as heard over the chest-wall corre-

sponding to the surface of the healthy lung. This consists of a mere humming or buzzing, in which all trace of articulation is lost.

We have already indicated that there are individual peculiarities of vocal resonance audible over healthy chests, for which allowance must be made, and of which a just estimation can be arrived at only by experience. Moreover, on the right side the vocal resonance is as a rule stronger than on the left. This fact diminishes the significance of an exaggerated resonance on the right side, while it enhances the importance of a like change on the left side. We have never to depend upon changes in vocal resonance alone for a diagnosis. When such changes result from diseased conditions, other signs will not be wanting in the breath sounds, or on percussion.

Morbid Alterations of Vocal Resonance over the Chest.

1. *Simple Increase* (*a*) and *Diminution* (*b*) of vocal resonance (ascertained by comparison).

2. *Bronchophony* transmitted in greater or less perfection, and audible over a portion or portions of the chest-wall which correspond anatomically to lung substance.

1 (*a*) *Simple Increase of Vocal Resonance.*—This occurs in partial consolidations of the lung as in tubercular infiltrations, where the sign is chiefly useful. The natural differences existing between the two sides in this respect must, of course, be borne in mind, as well the fact that over the upper portions of the lungs vocal resonance is stronger than over the lower. Simple increase passes insensibly into "bronchophony" by augmentation of the intensity and concentration of the sound.

(*b*) *Diminution of Vocal Resonance*, down to absence of the same, occurs in pleuritic effusion, and occasionally in consolidation of the lung, when probably the bronchi passing through the affected structure are blocked with secretion. Usually consolidation gives rise to bronchophony; but we must not forget that where all the conditions which usually favour

transmission of the voice sounds (and breath sounds) seem to be present, vocal resonance may be inexplicably diminished or even absent. In pneumonia, we know how unaccountably the tubular breath sound may appear and disappear from time to time. Diminution of vocal resonance occurs where there is pressure from without on one of the main bronchi, as by aneurism or tumour. It is met with in cases of chronic fibroid consolidation of the lung, where the pleura is greatly thickened and the bronchi are blocked with secretion.

2. *Bronchophony.*—Under certain abnormal conditions, the vocal resonance in the form we have described it as audible over the thoracic walls corresponding to lung parenchyma, is replaced by a voice similar, if not identical, with that heard over the trachea or bronchi. The conditions which accomplish this abnormal transmission and perhaps reinforcement of the voice, are those we have already mentioned as being productive of a breath sound of bronchial type audible over portions of the chest, where in health there should be heard vesicular breath sound. They are as follows:—Conditions in which the bronchial tubes passing through the lung are surrounded by solid, in place of air-containing structure, or the presence of excavations, *in already consolidated tissue*, which freely communicate with bronchi, or again of dilatations of the bronchi themselves, *provided that their walls have become thickened and the surrounding parenchyma consolidated to a certain depth.* Various diseases furnish these conditions. Pneumonia, phthisis, and pleurisy with effusion, do so most commonly. The last acts, of course, by compressing the air out of the lung, and thus condensing its substance around the bronchial tubes. The lung in such cases is pressed backwards, and bronchophony is best or alone audible posteriorly, especially towards the root where the bronchi enter. Bronchophony, when it occurs over pleuritic effusion, is usually weak, but the voice possesses a clearness which stamps it at once as "bronchophony"; just as the "ch" quality of the breath sound heard under similar circumstances, however feeble the murmur itself, leaves no doubt in the mind of the practised

auscultator, as to the nature of the breath sound. Growths in the lung sometimes produce the necessary conditions for bronchophony, intervening between patent bronchi and the surface. As already indicated, bronchial breath sound usually accompanies bronchophony, and not unfrequently in pneumonia it is heard as a "whiff" after each word. Obstruction in the bronchial tubes, whether produced by pressure from without or by plugging of the lumina with secretion, interferes with and may annul bronchophony when the conditions are otherwise favourable to its occurrence.

The most exaggerated forms of bronchophony are met with over cavities surrounded by consolidated tissue, possessing smooth walls freely communicating with a large bronchus, and in close approximation with adhesion to the chest-wall. In such cases the voice seems to pass directly into the ear of the auscultator, and its distinctness and articulation remain unimpaired, if not enhanced, during whispering. This is the so-called pectoriloquy about which so much has been written. Laennec seems to have been first struck with the occurrence of the voice, as thus abnormally audible over the thorax, and then from theoretical considerations to have listened for a similar sound over the larynx and trachea in health. He says,—"I presumed consequently that an analogous phenomenon ought to be found on applying the stethoscope over the larynx and trachea of a healthy man. My conjecture proved correct . . . . . This experiment is a good means of obtaining an exact idea of pectoriloquy, when patients are not at one's disposal." Unfortunately for diagnostic purposes, this perfect transmission of the voice sound can occur in mere hepatization of the lung, and apart from the presence of cavities, other than those of the bronchial tubes passing through the solid organ. When a cavity is large and has smooth walls, while it freely communicates with a bronchus, the voice, as other sounds, may acquire amphoric echo. This is a reliable sign of a large cavity, but it is by no means common. Cavities surrounded by healthy lung tissue would probably have little effect upon the condition of the voice.

No description of the voice phenomena in relation to the auscultation of the chest would be complete without mention of ægophony. "Laennec's Ægophony" it is often called, although nearly all the physical signs we describe may equally well bear his name. Laennec did, however, attach a special importance to the sign, for he devoted a disproportionate space of his *Traité de l'Auscultation Médiate* to its consideration. He did not regard it merely as a modification of bronchophony, inasmuch as he speaks of its occurrence along with the latter sign. We believe it best to quote the author's own description. "Simple ægophony consists of a peculiar resonance of the voice, which accompanies or follows the articulation of the words. It seems as if a voice more shrill, sharper than that of the patient, quivered at the surface of the lung. It appears to be rather an echo of the voice than the voice itself. It seldom rises into the stethoscope, and hardly ever passes through it completely. It has besides a constant character, after which, I have thought it right to name the phenomenon (goat-sound): it is tremulous and jerky, like the bleating of a goat, and its tone, according to the description we have just given, comes equally near to the voice of the same animal." Notwithstanding Laennec's belief that ægophony was something distinct from bronchophony, we cannot regard it as more than a modification of the latter. Apart from the conditions which give rise to bronchophony—usually a weak bronchophony—we know nothing of the sign. Laennec himself admits that he long confused it with bronchophony. The locality of the sign is a somewhat limited one; it occurs in the neighbourhood of the inferior angle of the scapula, usually extending a few inches to both sides of this point, and it seldom extends as far forwards as the nipple, at the level indicated. Laennec held that this sign was always the result of fluid in the pleura, and he imagined that the bronchi became flattened by pressure for its production, and that during the acme of effusion it disappeared owing to obliteration of the lumina of these tubes. His explanation of the mode of production is certainly erroneous; but if we consider the sign only in its perfect develop-

ment and limited to the locality mentioned, while we make allowance for the fact that individual voices possess a tremulous character, which, when the voice is rendered bronchophonic by disease, unaccompanied by effusion, will closely simulate true ægophony, the bulk of evidence seems in favour of Laennec's view, namely, that pleural effusion in moderate amount is necessary for the production of ægophony. In cases of pleuritic effusion, where Laennec had heard ægophony widely distributed, he found the lung partially adherent to the chest-wall, and thus prevented from retracting completely. It is probable that consolidation, along with a thin layer of pleuritic effusion, may produce ægophony of wide range. The great question is whether solidification of the lung can alone produce the sign. We are contented to leave it unanswered, believing it to be of trifling importance. The presence of fluid can always be ascertained by aspiration with a hypodermic syringe, and a careful survey of all the information at our disposal will seldom render resort to this procedure necessary.

SUCCUSSION.

This method of physical diagnosis consists in gently shaking the patient's trunk while the ear is applied to its surface. The sign to be elicited by it, though applicable in comparatively rare conditions, is remarkable for its antiquity, having been referred to by Hippocrates. It is consequently often spoken of as Hippocratic Succussion Sound. The condition necessary for the production of this sound is the presence of *air and fluid* in a large cavity. On shaking the patient, splashing sound, with which every one is familiar, results, and is accompanied, owing to its production within a large echoing cavity, by amphoric resonance (described in another place). Where we hear this sign, it affords us certain information of the existence within the chest of a large cavity containing air and fluid. Such cavity must either be an enormous excavation in the lung or the pleural sac. In the great majority of cases, it is the latter. It has been stated that hydro-pneumothorax furnishes the sound in greater perfection than pyo-pneumothorax, in which the fluid is thicker.

The possibility of mistaking a splashing sound produced in the stomach for left pleural or pulmonary succussion sound has only to be mentioned.

APPENDIX ON LARYNGOSCOPY.

It is important that the student of pulmonary diagnosis should be familiar with the appearance of the larynx as revealed in the laryngoscopic mirror. The following diagrams are taken from Dr. Prosser James' *Lessons in Laryngoscopy*, and show the larynx with the cords respectively, in the state of abduction as during a deep inspiration, and in that of approximation or adduction. We consider, firstly, the structures forming the

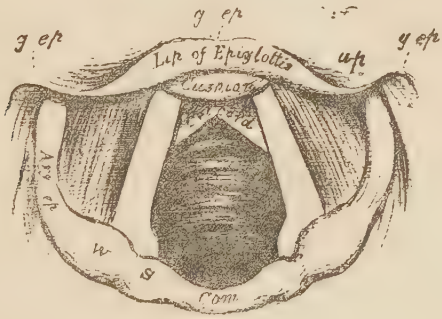
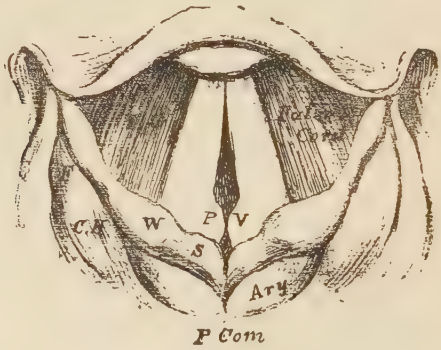


Fig. 1.

circumference of the larynx. Uppermost in the mirror is seen the epiglottis, which varies much in shape in different individuals. The superior surface is of a dirty pinkish colour; the lip is yellow, with a slight pinkish tinge, and when the edge only is visible, it appears as an almost white line. The projection on the under surface seen below the lip is named the cushion, and is of a bright red colour. When a larger portion

of this surface is visible, it also appears bright red. The lateral boundaries of the larynx are formed by the arytæno-epiglottidean folds (*Ary. ep.*) which stretch from the epiglottis to the arytænoid cartilages. The latter cartilages are surmounted by the small cartilages of Santorini (*S*). In the arytæno-epiglottidean folds, to the outer side and in close proximity to the cartilages of Santorini, are situated the cartilages of Wrisberg (*W*). These vary in shape and size, and sometimes small cartilaginous nodules are interposed between them and the cartilages of Santorini.

Right.



Left.

Fig. 2.

The definition of the projections in the arytæno-epiglottidean fold, due to the cartilages named, depends not only upon the shape and size of the cartilages themselves, but also upon the thickness of their covering. The colour of the arytæno-epiglottidean folds has been justly compared to that of the gums, but over the cartilages of Santorini and Wrisberg it is of a somewhat brighter red than elsewhere. Between the arytænoids is the posterior commissure (*Com.*), which folds upon itself when the cords are approximated, as shown in Fig. 2. Outside the arytæno-epiglottidean fold is the small fossa, sometimes termed hyoid, under the floor of which the great cornu of the hyoid bone is situated (*C.H.*)

Of the parts situated within the boundary we have just described, the most conspicuous objects are the true vocal cords, to which attention is attracted, not only by their pearly-white colour, but by their movements during respiration, and still more during phonation. They pass from the angle of the thyroid to the anterior or vocal process of the arytænoid cartilages. The latter point of union is spoken of as the vocal process (*P. V.*), in front of which the glottis is inter-ligamentous, and behind which it is inter-cartilaginous. When these processes are rotated outwards the glottis becomes lozenge-shaped, as in Fig. 1. Above the true cords are seen the so-called false cords or ventricular bands. They are of a deeper colour than the arytæno-epiglottidean folds, and the openings of the ventricles may be sometimes seen as "dark lines" between them, and the true cords beneath. "When the vocal cords are approximated, a small depression may be seen near the epiglottis between the ventricular bands below and the ary-epiglottic folds above," which Dr. Mackenzie has proposed to call the fossa innominata. In front, below and between the vocal cords, the cricoid cartilage may be recognised, and still lower the rings of the trachea. In exceptional cases, the bifurcation of the trachea is seen.

We can here only point out in what direction abnormities are to be looked for, and must refer the reader to special works on the subject for details.

The larynx may be simply pale and anæmic, a condition which has been said to indicate a predisposition to phthisis. The opposite condition, undue redness from hyperæmia, is more important; it may be generally distributed over the larynx, or limited to parts. When the cords are affected they lose their pearly whiteness, and become more or less red. From simple hyperæmia to alteration in the form of parts due to inflammatory œdema, is but a step, and œdema of the larynx constitutes an important feature in many of its diseases. The arytæno-epiglottidean folds are found greatly swelled and semi-transparent, looking like blisters when œdema is present. In the chronic

laryngitis of phthisis, these folds are apt to become much enlarged, chiefly from this cause, the bases of the pyriform tumours into which they are transformed being placed posteriorly at the arytænoids. Inflammatory swelling may be limited to parts, or pretty generally distributed. New growths not connected with inflammatory processes are common in the larynx, especially papillomata. They are usually easy of recognition by means of the laryngoscope. Lastly, there may be loss of substance, varying from a superficial abrasion to deep ulcers in connection with necrosed cartilages. The appearance of exudation or secretion lying on the surface of the mucous membrane can usually be referred to its true cause.

We have next to consider abnormities in the motion of the vocal cords, in the detection of which the laryngoscope has rendered so much service to medicine. The simplest form of paralysis is the unilateral, where the unaffected side is available for comparison. Paralysis occurs in the great majority of cases on the left side, being due to thoracic aneurisms or other tumours pressing upon the left recurrent laryngeal nerve. The larynx in such cases is as a rule quite healthy as regards colour and form. Where there are the evidences of local change, caution is necessary in drawing a conclusion as to the cause of the paralysis, as it may be merely the result of local inflammation. When the recurrent laryngeal nerve is paralysed, the vocal cord of the affected side is observed to be quite motionless during respiration and phonation. The adduction of the unaffected cord is frequently exaggerated during the latter, so that it passes the middle line to meet its fellow. Thus the voice may remain well preserved, and the arytænoid cartilage of the unaffected side seems to pass in front of that of the other side, giving an appearance of obliquity to the image. Slight approximation of the arytænoid cartilages may still, however, be accomplished, for not only must the unaffected half of the arytænoid muscle act on the arytænoid cartilage of the paralysed side, but this muscle derives a special nervous supply through the superior laryngeal nerve. In practice it occasionally happens that only

the arytænoid cartilages can be seen, the epiglottis hiding the rest of the larynx; in such a case it is not safe to conclude that there is no paralysis of the cords because there is some approximation of the arytænoids. Adduction of both cords may be perfectly performed during phonation, but one cord appears to give way and become curved, while the other remains straight. This probably depends upon paralysis of the crico-thyroid muscle, which is supplied not by the recurrent laryngeal, as the other muscles of the larynx, but by the superior laryngeal nerve. The affection has only importance as a cause of hoarseness.

Bilateral paralysis of the muscles of the larynx may affect the abductors or adductors of the vocal cords, or both sets together. The two varieties last named are common in hysteria, being the usual cause of hysterical aphonia. Paresis rather than paralysis is, however, the condition generally present. When both abductors and adductors are paralysed, there is superadded to aphonia, occasional stridor when a deep inspiration is made. Paralysis of the abductors alone, is an important affection seriously embarrassing respiration. It by no means always owns a hysterical origin. It must be recollected that the floor of the ventricles of the larynx is concave. When the cords are approximated during inspiration, as they must be if the abductors are paralysed, the column of air will bear directly upon the floors of the ventricles just external to the true cords, which will thus tend to become approximated after the manner of valves. In such cases the voice is unaffected, but there is inspiratory stridor whenever a deep breath is taken or any exertion is made, as well as frequently during sleep. The condition is easily recognised by the laryngoscope when the cords are found to keep close together and even to approach one another during inspiration, instead of being drawn apart as in health. On phonation nothing abnormal is observed. Paralysis of the abductors or adductors alone is probably of central origin.

Spasm of the adductor muscles of the cords occurs after the application of any irritant to the larynx, causing the well-known inspiratory crowing noise which follows such application. It

produces the "hoop" of hooping-cough. In the inflammatory affections of the larynx, it is much more likely that paralysis or paresis rather than spasm is induced, and that the apparently spasmodic signs are really dependent on the cords not being sufficiently abducted during inspiration to prevent the valvular action we have referred to, taking place.

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